PNEUMONIA AND PNEUMO-COCCUS INFECTIONS

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PNEUMONIA.

CHAPTER I.

INTRODUCTION.

As time has gone on the ideas associated with the word pneumonia have undergone many changes. They have become progressively more and more definite and as our knowledge of pathology and our diagnostic resources have increased, the number of conditions included have been further and further reduced until now the term is employed only in connection with acute infections of the lungs associated with consolidation of these organs. In this little monograph the word is still further restricted and whenever the word pneumonia is employed it means acute inflammation of the lungs with exudation in the alveoli as the result of infection with the diplococcus pneumoniæ of Fraenkel-Weichselbaum. Very similar clinical and pathological pictures result from infection of the lungs with other organisms, but all of these are relatively so infrequent as compared with the pneumococcus pneumonia that they may be neglected, and they will be referred to here only in the effort to outline the features which will enable one to distinguish them clinically from the pneumococcus cases.

This subdivision of the general clinical picture of pneumonia into subgroups with the causal organ-

ism as the basis of classification is now justified by the fact that it is usually possible to determine clinically the organism at work and whenever any disease can be traced back to its ultimate cause, our knowledge is clarified, our prognoses become more definite and our therapeutics more rational. This seems self-evident but may gain force by examples. No one, who today is practicing medicine as it should be practiced, will neglect discovering what form of the malarial organism is the cause of each particular case. In this instance not so much because the treatment is altered by the exact knowledge of the cause as that the prognosis becomes more definite. Every effort should be made in every case of throat infection, pharyngitis or tonsillitis, to ascertain the causal organism, not alone because the prognosis of the possible nature of complications and sequelæ is influenced by this knowledge, but also and particularly because the treatment is thereby determined. So also with pneumonia, a diagnosis of pneumonia is no longer sufficient. The causal organism must be sought and the diagnosis must run, pneumonia due to the Friedlander bacillus, the influenza bacillus, the tubercle or typhoid bacillus, the pneumococcus, et cetera. It is to be regretted that this knowledge does not as yet determine the treatment but it has a great influence upon the prognosis, a fact which will be keenly appreciated by anyone who has ever mistaken an acute pneumonia due to the tubercle bacillus for one due to the pneumococcus or vice versa.

It is reasonable to hope that at some time in the future, and may that time be near, such exactness of

diagnosis will determine the treatment. It is reasonable to expect that some fair day a specific treatment for pneumonia will be discovered as efficient as the antitoxin in diphtheria, but the employment of a specific therapy presupposes a specific diagnosis.

Another and important fact which recent years has made clear is that the pneumococcus is capable of causing other clinical pictures than that of the pneumonia and it seems reasonable to include here some, even though brief, description of them. With this introduction we can take up the subject of pneumococcus infections, particularly the pneumococcus pneumonia, but before doing so it would be well to dwell upon a fact now very generally known, namely that in this part of the world pneumonia has come to be one of the most important of the problems confronting the profession. Laennec, whose genius shed so much light upon this and other diseases of the thoracic viscera says of pneumonia: "This is one of the most serious and the commonest diseases and in frigid and temperate climates causes more deaths than any other acute disease." Juergensen thirty years ago said of it, "Next to phthisis it is one of the most dangerous enemies of man." Since then the question has still further altered and today pneumonia causes more deaths than tuberculosis.

The growing importance of the disease is everywhere attracting serious attention but nowhere has more thought been put upon it than by the Health Department of Chicago. In the Bulletin from this department dated January 17, 1900, we find the

statement that pneumonia is the cause of one-eighth of all deaths and that it caused 46 per cent more deaths than all other contagious diseases combined. The deaths from tuberculosis are exceeded by one-third. Between the years 1860 and 1900 pneumonia has increased from 4.4 per 10,000 to 19.95 per 10,000.

If any one were asked to name the three greatest problems now engaging the attention of the medical profession, all would agree on tuberculosis, pneumonia and carcinoma and because of their great frequency the first two are the most important. I think, too, that we may be fairly well satisfied with the progress being made in handling the tuberculosis problem. We know much of the methods of prevention and are doing fairly well in the treatment of the cases. For illustration we may again quote the Chicago Health Department which finds that between 1860 and 1890 the deaths from tuberculosis per 10,000 decreased from 25.28 to 15.30. This reduction is encouraging and were complete use made of the knowledge in our possession tuberculosis could be reduced to a rarity. If the general public could be brought to an appreciation of the importance of learning early the cause of any cough and the profession would employ to the full its aids in the early diagnosis of tuberculosis, cases beyond the point at which treatment ceases to be of benefit would be rare. If these conditions were fulfilled, and they are not impossible, the proper care of the sputum would naturally follow, the milk supply would be properly looked after and even within one

decade tuberculosis would cease to be a serious problem.

If one compared today the relative importance to the State of tuberculosis and pneumonia, the former must be considered the more important. Tuberculosis after causing a considerable period of partial disability, makes its victim an invalid for months, thus destroying his earning capacity and yet leaving him an expensive burden for the State to carry. Moreover, its victims are most numerous among the most valuable members of society, the young adult. Just at the time when the State is beginning to realize upon the investment made in rearing and educating the individual, he becomes a victim of a lingering and expensive illness.

From this point of view tuberculosis is the more important disease of the two, but when we recall that pneumonia is steadily and rapidly increasing in frequency, that while our knowledge of the pathology and symptomatology is growing daily, our methods of treatment and prevention are at a standstill, then it becomes clear that for the profession this problem is the greater of the two. The treatment of pneumonia is today as unsatisfactory as it was two hundred years ago, and when we are asked for methods of prevention of the spread of this disease we answer in the most general terms, knowing fully that we are asked for something which we have not.

CHAPTER II.

PATHOLOGY.

Pathological Anatomy. Since the days of Laennec, pathologists have recognized, as he did three stages in the course of a croupous pneumonia and have and still employ the terms applied by him to them, namely, a stage of congestion or engorgement, a stage of red hepatization and lastly a stage of gray hepatization. To these three we now add a fourth stage, one of resolution. While these stages are well marked in the lungs they do not correspond to any variation in the clinical picture and therefore have only an indirect clinical interest, and it is impossible to say from clinical evidence what stage the pathologist will find on examination except that if death occurs in the first 24 to 36 hours the first stage, that of engorgement, will be found, the next two or three days will probably show red hepatization and the next three to five days will show the gray hepatization. Generally, however, all three stages will be found in different parts of the same lobe or in different lobes.

Inasmuch as death does not often occur within so short a period as one or two days after the onset of the disease it is not often that one sees the congestive stage pure and simple and material for the study of this stage must be sought in the borders of the pneu-

monic area. When this stage is found the affected portion of the lung is dark and bluish in color. It is firmer and heavier than normal and the imprint of the fingers remains in the lung much as it does in an edematous leg. The lung still crepitates, for the air is not yet entirely excluded from the alveoli, although one must infer from the dark color that the oxygenation of the blood in the affected area is much reduced. The dark blue color is in sharp contrast with the red or pinkish color of the healthy parts of the lungs and this contrast is heightened by the fact that it does not collapse to the degree a normal lung does. Upon section the lung is found to contain a greatly increased amount of fluid. This fluid is deeply blood-stained, slightly frothy, and runs freely from the section. The lung tissue is much more easily torn than it is under normal conditions.

Without a sharp line of division this condition passes over into that of red hepatization. The color is less livid but remains a dark red. There is absolutely no collapse upon opening the thorax. The affected portion is firm and does not crepitate at all. It is heavier and sinks at once and rapidly when thrown into the water while in the congestive stage the lung still floats but floats low.

When the consolidation is complete the specific gravity of the lung bears to the normal a relation of 19:1 according to Genarin. The weight is greatly increased. Eichorst found a consolidated left lung weighing 1500 grams more than the right lung, but generally the increase is not so great, ranging from about 300 grams upward. The normal weight of

both lungs ranges from 950 to 1500 grams, the right being a little the heavier and the specific gravity varies from 0.345 to 0.746. Upon section the deeper parts of the lungs are found to be a dark red color. The amount of fluid is less than that obtained on section during the first stage and it is thicker and not frothy. The tissue has become so friable that very light pressure with the fingers is sufficient to tear it. The surface of the section is granular and if the knife is scraped across the surface these granules are detached. One also finds in the scraping very small, yellowish branching particles, which are the masses of exudate present in finer bronchi just as the granules are from the alveoli. The size of these granules varies with the age of the patient. In children they are small, being, according to Damaschino, who laboriously measured them, from 0.07-0.11 mm. In adults they vary from 0.17 to 0.21 mm., thus corresponding in size to the normal distended alveolus. In the aged the granules are yet larger, ranging up to 0.27 mm. In cases of emphysema they may be still larger.

The question whether a lobe or lung is increased over its size at full inspiration, by all this exudate in its alveoli has been the subject of argument, but there seems to me several good reasons for believing that there is an actual increase in size. Not only are the alveoli distended with exudate, but the blood vessels also are over filled, the pulmonary tissue is infiltrated with fluid, all of which together would make the lobe larger than normal. In many cases the imprint of the ribs is found on the infiltrated area indicating pressure against them greater than

is normally present. There is also clinical evidence that the lobes are increased in size. I have in favorable cases, i. e., cases where the pneumonia was confined to one lobe and the pleura free from any considerable exudate, been able to demonstrate that the outlines of the dulness, while corresponding accurately to the shape of the lobe in question, did not in size, being in some instances as much as 2 centimeters larger. A second bit of clinical evidence is found in the slight cardiac displacement occurring in some cases of pneumonia just as it frequently does with pleurisy.

The stage of gray hepatization resembles the second stage in many ways. So far as size, consistence, weight, specific gravity and friability are concerned, there is little difference, but the red color is replaced by a pale or gray yellow.

The fourth stage, that of resolution, looks in many ways like the gray hepatization, but the lung is softer, the granular appearance on section is entirely gone and the fluid scraped from the section is thinner, more abundant and purulent.

The rapidity with which these stages pass from one into the other cannot be correctly estimated, but there are good clinical reasons for believing that they may do so with great rapidity. A pneumonia presenting all of the physical signs of a complete consolidation sometimes develops in 24 hours and presumably goes through the first and second stages in doing so. Much more frequently all the signs of consolidation will disappear in an equally short period and presumably go through the third and fourth stage in doing so. This would make but 48

hours for the entire circle of changes, so short a time as to seem impossible.

The microscopical appearances of the lungs vary as greatly as does the gross appearance, according to the age of the process. For a long time it was believed that the first change consisted in the distention and over-filling of the pulmonary capillaries, but later studies partly of an experimental nature on animals and partly of specimens obtained very early in the course of pneumonia have led to the opinion that the primary change is in the epithelial lining of the alveoli. These cells become swollen, granular and the nuclei undergo division. Many are detached and become rolled together in a mass in the lumen of the alveolus. Together with these are found considerable fibrin surrounding the epithelial cells and in addition some red blood cells. The fibrin is found not only in the lumen of the alveoli but extends also through the alveolar walls, thus making a connection between neighboring alveoli.

The fibrin is no doubt derived from the blood rather than from altered epithelium, as some have assumed, and the escape of the fibrin from the blood is favored by and is secondary to the changes in the alveolar epithelium. These changes deprive the capillaries of the support to which they are entitled and they become greatly distended. The transudation of the fluid parts of the blood increases and in addition to this the red corpuscles escape freely, so that as Aufrecht says "the second stage, the red hepatization, is not the result of exudation, but is due to the coagulation of blood which has escaped from ruptured capillaries." The hemorrhagic char-

acter of the process is shown by the early appearance of blood in the sputum and in this stage of red hepatization the process may be compared with the formation of a thrombus in a ruptured blood vessel.

With the completion of these changes begins the migration of the white blood corpuscle which marks the beginning of the gray hepatization and is its most essential feature. The red corpuscles gradually disappear and the changes which take place in their coloring material are largely responsible for the changes in color from red to gray. The white corpuscles penetrate the network of fibrin. During the periods of red and gray hepatization the alveolar epithelium does not undergo any further change. With the fourth stage the exudate becomes granular and undergoes absorption. Only a small portion of the exudate is expectorated.

The formation of fibrinous masses is not confined to the alveoli, but extends also outward into the finer bronchi. These bronchial plugs, as has been mentioned, can be obtained in the shape of dichotomously dividing whitish threads on scraping the knife over the surface of the cut lung. They may also be obtained in the sputum and constitute an important peculiarity of the sputum of pneumonic patients. In general these bronchial thrombi do not extend into any but the finest bronchi, but in some cases, and I should say that this is more common than is generally supposed, they extend into and plug even large bronchi. This fact has a very important clinical bearing because of the effect which it has upon the physical signs, making them resemble those of a pleural exudate by destroying the increased

vocal fremitus, the bronchial breath and voice sounds.

In practically every case of pneumonia, excepting only those rare instances in which it remains central, the pleura covering the affected lobe undergoes changes. The smooth, glistening surface is roughened, the blood vessels are distended and often ruptured, causing punctiform hemorrhages. A layer of loosely attached fibrin forms over the surface. The thickness of this layer varies greatly. It is often no thicker than tissue paper but ranges up to a half inch or more. It may be firm or quite loose in its texture. The amount of fluid exuded into the pleural cavity varies from none at all up to several hundred c. c. It is scarcely necessary to mention the fact that such exudates alter the physical signs of the pneumonia often to such a degree that those of the pleurisy will entirely obscure those of the pneumonia. This, however, is a matter to be taken up in detail in the clinical chapters.

The bronchial glands are found swollen and infiltrated, sometimes softened and purulent. They usually cause no particular clinical disturbance, but that they may do so should be kept in mind and will sooner or later be found to explain some things which otherwise would be difficult to understand.

The right side of the heart is often found dilated and full of blood while the left ventricle is empty. The distention of the right heart is partly the result of mechanical conditions, the obstruction to the flow of blood through the lungs and partly due to changes in the heart muscle. This is often found cloudy, pale and soft and under the miscroscope the muscle fibres

are granular and some of them fatty. Cagnetto examined the myocardium in 8 cases of pneumonia dying within two weeks of the onset of the disease, and found a marked acute myocarditis in 4 of them. The muscle fibres were homogeneous, the nuclei altered in shape and staining reaction and in some places the muscle fibres were entirely destroyed and replaced by young connective tissue. Many of the capillaries and small arteries showed an acute endarteritis. It seems probable from certain clinical data that the changes in the heart muscle are more common than is generally supposed. The various authorities on pathology, while giving much space to the changes in the lungs, dismiss the heart with a word or two and yet clinicians, paradoxical as it may sound, are more concerned with the condition of the heart than of the lungs. The kidneys are usually somewhat swollen, hyperæmic and the parenchyma cloudy. The changes rarely pass beyond the stage of cloudy swelling, but may become intense enough to amount to actual nephritis.

Changes in the other organs are rather in the nature of complications and will be mentioned in connection with the clinical phenomena corresponding to them.

CHAPTER III.

ETIOLOGY.

Etiology. The diplococcus of pneumonia was first discovered by Sternberg in his own sputum in 1880, but its relation to pneumonia was not suggested by him nor by Pasteur in whose laboratory the same organism was found in the saliva of a child dying of rabies in 1881. Even thus early Pasteur knew that this organism was pathogenic to rabbits, causing a rapidly fatal septicæmia. He knew also that the bacterium became rapidly attenuated and that rabbits inoculated with bacteria of lesser virulence became immune to the action of others of high virulence. The relation of this organism to pneumonia was worked out especially by Fraenkel and Weichselbaum, after whom the organism is named. The French claim this for Talamon and usually speak of it as the pneumococcus of Talamon-Fraenkel. Sternberg also is a claimant for the honor and in this country at least there is a disposition to grant his claim. Fraenkel in 1883 obtained from a pneumonic lung a lancet-shaped diplococcus which was pathogenic to rabbits and multiplied in their blood. It caused pleurisy and pericarditis and sometimes a pneumonia exactly similar to that of man. Weichselbaum in 129 cases found the diplococcus pneumoniæ 94 times in the lungs. He found it also

in the complicating pleurisy, pericarditis, peritonitis, meningitis, endocarditis and arthritis. Wolff found this same diplococcus in 66 of 70 cases. These results have since been confirmed by many different observers many, many times and while all agree today that the clinical picture called pneumonia may result from a variety of organisms it is in the great majority of cases due to the diplococcus pneumoniæ. This belief has been further strengthened by the recent work done in the way of cultures taken from the blood during life. The technique of this method of taking cultures has been so perfected that the diplococcus has been found in something over 92 per cent of cases clinically recognized as pneumonia. This is considerably higher than the percentage in Weichselbaum's report of the post mortem examination. He found 94 in 129 cases, i. e., 73 per cent. The organism is easily found in the sputum of pneumonia patients. It stains readily with the various aniline dyes and by Gram's method. The sputum is spread upon a slide in the ordinary way, dried, and then stained with any aniline solution. In the sputum the cocci are usually arranged in pairs, but sometimes in short chains, and are surrounded by a distinct and relatively thick capsule. The shape is lancet-like and from this comes the name micrococcus lanceolatus, often applied to it.

The coccus grows well upon blood serum, best at 37° centigrade, in the shape of very fine, colorless colonies. It grows also in bouillon, which is the medium usually employed in making blood cultures. The organism is pathogenic to mice, rabbits and guinea-pigs causing a rapidly fatal septicæmia which

may or may not be accompanied by a pneumonia. The organisms rapidly lose their virulence in cultures.

It was known also even as early as 1885 when Weichselbaum made his report, that this organism excited other clinical pictures than the pneumonia, but the importance of this fact and the frequency and great diversity of the cases due to this organism is not yet as generally appreciated as it should be. They differ as greatly from each other and from the common clinical picture of pneumonia as cases can differ, and yet diverse as they are, there are certain features present in all which suggest their relation to the pneumococcus and the examination of the blood for bacteria will in most of them prove that this organism is the causal factor.

While no one now questions the pathogenic relation of the diplococcus pneumoniæ to the great majority of the cases of croupous pneumonia, it is certain that there are other factors which are important in the etiology of this disease, preparing the soil for infection. This organism is singularly ubiquitous, being found everywhere throughout the world, a fact which is obvious from the universal distribution of pneumonia, but it is found also frequently in the throats of people who are not and never have been sick with pneumonia. Both Sternberg and Pasteur found the organism first in the saliva. Bein upon the basis of studies of the saliva of healthy individuals estimates that the diplococcus is found in about 50 per cent and other observers place the figure anywhere between 15 and 30 per cent. Von

Besser found it in the nasal secretion of 14 per cent of 57 people examined.

In view of these facts it would seem almost that this diplococcus is normally a harmless parasite comparable to the bacterium coli communis, but like the latter, capable of causing disease if certain conditions exist. What the nature of these other aiding factors is we do not know for certain, but a consideration of some of the influences which favor the onset of pneumonia will be profitable.

Let us consider first the frequency of pneumonia as compared with other diseases. This is not a constant figure for it varies from year to year and from month to month. On the average pneumonia makes up about 3 per cent of all illnesses. In Vienna it is placed at 2.6 per cent; in Berlin at 2.5 per cent. Townsend and Coolidge, in a review of 1,000 pneumonias from the records of the Massachusetts General Hospital, say that they make from 1 and a fraction to 3.5 per cent of the admissions. E. F. Wells, in 400.366 admissions to various hospitals of the world, found that the pneumonias made up between 2 and 2.5 per cent. Aufrecht found 1,501 cases of pneumonia in 36,540 admissions, which is 4.1 per cent. Juergensen found in the Polyclinic of Kiel 203 pneumonias in 3,993 patients, *i. e.,* 5.0 per cent.

If pneumonia is compared with internal diseases only the figures are generally higher. Juergensen places it at 6.4 per cent in Germany, England and France. In the 1900 census of the United States pneumonia makes practically 10 per cent of all deaths, i. e., it is the cause of about 10 per cent of

the serious illnesses. One cannot, however, make an estimate from these figures of the relative frequency of pneumonia and all other illnesses.

Wells gives the following table in which the figures are much lower:

	Internal Disease.	Pneumonia.	Per Cent.
January	1,576	31	2.0
February	1,999	58	3.0
March	2,765	60	2.2
April	3,908	57	1.4
May	3,280	33	1.0
June	4,006	23	0.5
July	6,256	14	0.2
August	5,431	17	0.3
September	2,394	8	0.3
October	1,459	20	1.4
November	1,251	34	2.8
December	1,384	41	3.0

The table shows also the influence of the time of the year upon the prevalence of pneumonia.

The fact that pneumonia is becoming relatively more common is most important and serious. This is very generally true throughout the world, but in no place is this increase more alarming than in Chicago. The accompanying diagram, based upon the report of the Chicago Health Department, shows an increase of 350 per cent in the pneumonia deaths in four decades. This is not the result of any increase in the severity of the disease, but to an increase in the number of cases. If the average mortality is placed at 20 per cent, these figures mean that in 1860 there were 88 cases of pneumonia per

10,000 people, while in 1900 there are 395.6 cases in the same number.

Upon the same diagram are placed for comparison the number of deaths per 10,000 due to tuberculosis, showing that this disease is decreasing in frequency.

French also has drawn attention to the increasing prevalence of pneumonia, using Massachusetts as an example. Between 1861 and 1871 the deaths from pneumonia formed 3 per cent of the deaths from all

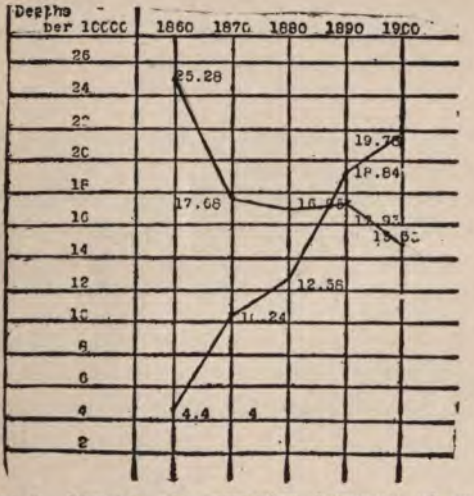


CHART I. Showing the increasing frequency of pneumonia and decreasing frequency of tuberculosis in Chicago.

causes. In the two succeeding decades it formed respectively 5 per cent and 6.7 per cent of the total number of deaths and in the last decade, 1891 to 1901, it rose to 10 per cent. That this is not peculiar to Massachusetts is shown by the figures already given from the 1900 census, 105,971 deaths from

pneumonia in a total of 1,039,094 deaths, *i. e.,* 10 per cent.

The influence of the time of the year upon the frequency of pneumonia is easily demonstrated by statistics and is admitted by all. Juergensen, from the statistics of Edinburg, Dublin, Copenhagen, Berlin, Zurich and Turin, puts 33.1 per cent of the cases in the winter quarter, 33.1 per cent in the spring, 16.1 per cent in the summer and 17.7 per cent in the fall, *i. e.,* 66.2 per cent in winter and spring, and 33.8 per cent in summer and fall. This is almost exactly the figure of Aufrecht based upon his 1,501 personal cases.

He had 69.3 per cent of his cases in winter and spring and 31 per cent in the summer and fall. Wells in combined reports of 685,566 fatal cases of pneumonia finds 70 per cent of them in the cold and 30 per cent in the warm months. In detail the cases are distributed as follows:

		Per Cent.	
December	65,667	9.6	
January	83,151	12.1	34.2
February	86,090	12.5	
March	89,062	13.0	
April	89,263	13.0	35.9
May	67,028	9.9	
June	38,861	5.7	
July	26,059	3.8	13.1
August	24,811	3.6	
September	27,183	4.0	
October	38,111	5.7	17.0
November	50,260	7.3	

The effect of season upon the frequency of pneumonia is better shown by the following diagram based upon 4,690 cases. It shows that the disease increases during the winter and reaches its maximum during March and April, months which are not so cold as earlier ones, but which are generally

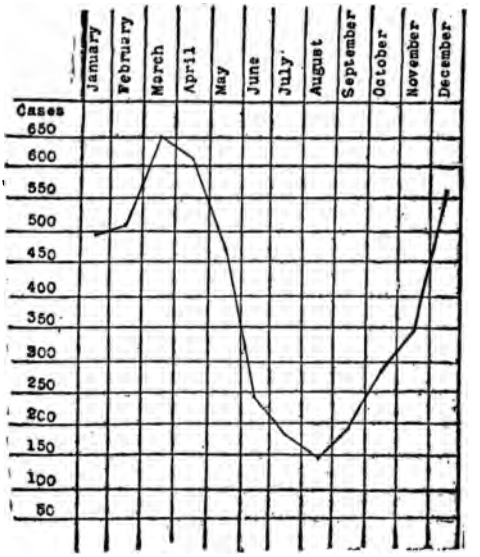


CHART J. Distribution of cases according to months.

regarded as more trying to the health because of the frequent changes in temperature and moisture and the boisterous winds. The minimum falls in July, August and September.

That the change in frequency is not due to mere temperature is supported also by the fact that pneumonia is not a common disease in certain countries

such as Iceland, where the temperature averages low, and is common in Arabia, India and Italy, where it ranges high. It seems rather to be a question of rapidly changing temperature than one of degree of temperature. Von Hirsch has drawn attention to the fact that the period of greatest frequency of pneumonia is not the same in all parts of the world, but in each place corresponds with that time of the year in which the temperature is most variable.

The influence of wind upon the prevalence of pneumonia has been frequently discussed, but it appears to me from a wrong standpoint. Wind is unquestionably an important factor in the diffusion of bacteria and if the dust which it carries is infected with pneumococci, frequent winds favor their spread. Wind, therefore, would be an important influence in a thickly populated district, especially if there is much dust, while it would have little influence under other circumstances. The amount of moisture and the barometric pressure have no known influence.

Before taking up the influences which affect the individual rather than the population as a whole something should be said of the contagiousness of pneumonia and its appearance in epidemics. When we recall the very general distribution of the pneumococcus and that it can be found in the air passages of all of us some of the time and some of us all the time, it is not necessary to assume contagion to account for any case. The daily experience in the hospitals of the world, all of which accept pneumonia cases and receive them into the general wards, is

strong proof against this disease being contagious. The development of a case of pneumonia in a hospital ward is very unusual. There are, however, occasional experiences which suggest strongly that under certain unknown circumstances the disease does become contagious. I have recently had occasion to mention a family living in a house which was to all appearances sanitary and satisfactory, in which three adults died of pneumonia in three days and one other member had died of the same disease a year previously. Schroeder speaks of a house in Kiel which yielded 25 cases of pneumonia in seven years. Keller mentions a house in Tübingen which furnished 8 cases in eight and a half years. Baduel and Gargani describe a house epidemic affecting eleven individuals. There are in the literature many reports of epidemics of pneumonia, but none of recent times, *i. e.,* during the period in which medical science had advanced to a point admitting of the circumscription now characterizing the word pneumonia, have been epidemics in the usual sense of the word. They have been house, ship, garrison or jail epidemics, endemics affecting numbers of individuals exposed to like circumstances, among which were some favorable to the development of the pneumococcus. The influences bearing directly upon the individual, which seem most important in creating a disposition toward pneumonia, are sex, age, occupation, previous attacks of pneumonia, habits and traumatism.

Any series of cases shows that pneumonia is much commoner in the male than in the female. This is illustrated by the following table:

	Males.	Per Cent.	Females.	Per Cent.
Aufrecht	1,223	81.4	278	18.6
Sears and Larrabee	714	75.2	235	24.8
Norris	382	76.4	118	23.6
Stevens	94	78.3	26	21.7
Townsend and Coolidge	724	72.4	276	27.6

If these figures are added together it shows *73.4* per cent males and *26.6* per cent females. Wells in 27,653 collected cases found 72.2 per cent males and 27.8 per cent females. The almost exact correspondence of the percentages derived from large series of cases and from widely distributed sources forces the suggestion that sex in some way has an influence upon the causation of pneumonia and that the women are relatively immune.

If one compares the relative numbers of the men and women entering a hospital with the number of pneumonias in each sex one gets figures which do not so closely correspond. Thus Juergensen says that 2.9 per cent of the men and 2.2 per cent of the women admitted to the General Hospital in Vienna between the years 1858 and 1870 were suffering from pneumonia. Aufrecht states that in the Alstädter Hospital in Magdeburg 4.1 per cent of all the patients have pneumonia, while 6.01 per cent of the men and 1.7 per cent of the women do. Thus the men are attacked three and one-half times as often as the women. Wells states that in 400,366 admissions to various hospitals pneumonia was the diagnosis in 2.5 per cent of the males and 2.0 per

cent of the females, almost exactly the figures quoted from the Vienna Hospital.

Mortality statistics do not serve as much of a guide to the frequency of a disease, but the 1900 census of the United States shows 58,340 males and 47,631 females dying of this disease. If the sex itself were the important factor in determining this difference in susceptibility to infection with the pneumococcus, the difference should hold for all ages, but in children under ten, pneumonia is as common in girls as in boys. There seems no good reason to question the current opinion that it is not the sex but the difference in the occupation and habits which causes the different susceptibility.

The influence of age may also be partly due to the difference in occupations and exposures. That age has an influence is clear from the following table:

Age.	Norris.	Aufrecht.	Sears & Larrabee.	Townsend & Coolidge.	Totals.	Percents.
1-10....	71	103	0	10	184	4.76
10-20....	71	333	66	132	602	15.6
20-30....	136	437	229	373	1,175	30.44
30-40....	84	259	241	217	801	20.75
40-50....	67	195	191	136	589	15.26
50-60....	30	83	93	80	286	7.4
60 up....	20	91	67	44	222	5.75
Total					3,859	

The influence of age is shown more strikingly in the following diagram based upon the material in the table, the curve being drawn on the basis of the percentages. The curve is still more striking if drawn with the absolute number of cases as a basis.

It is obvious that certain decades supply an undue proportion of the cases of pneumonia. Something over 50 per cent of the cases are between the ages of twenty and forty and over 80 per cent are from the forty years between the ages of ten and fifty.

There is a tradition to the effect that pneumonia is particularly prone to attack those who are strong

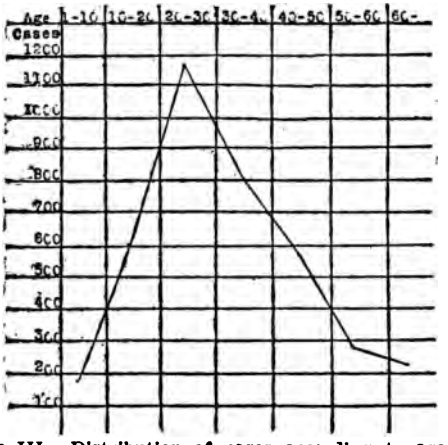


CHART III. Distribution of cases according to age.

and in good health, but this like many other dicta of the same sort does not stand any critical examination. It comes to us from a time when because of the ignorance of means of physical examination the terminal pneumonia of those debilitated by disease or confinement in jails and institutions passed unrecognized. Pneumonia in such people does not develop suddenly with the sharp and striking symptoms which characterize this disease in the vigorous and without the use of methods of physical examination the presence of pneumonia is not suspected. It seems probable, therefore, that either Dietl's figures were from a very special lot of material or he was prejudiced. Dietl in 750 cases of pneumonia found only 18 per cent who were previously entirely well.

More recent figures are much less surprising. Thus Chomel in 171 cases found 87 who were previously strong, 37 of average strength and 13 weak. Boulland found 20 of 26 cases strong and the others weak. Wells in 498 cases found 134 strong, 267 average and 97 weak. It is easy to see how the character of one's material would influence the relative numbers of the strong and the weak. No doubt the strong and healthy are frequently attacked because

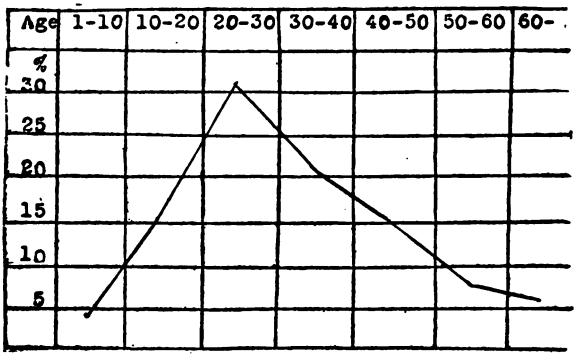


CHART IV. Percentage of distribution according to age.

of the exposure to which they are subjected, but with this as with other forms of infection, debility of whatever cause makes invasion by bacteria easier.

Authors very generally admit that occupation in some way influences the susceptibility of individuals, but our knowledge of this influence does not admit of any further classification of occupation than into indoor and outdoor. The latter contributes an undue number of cases of pneumonia, from 60 to 70 per cent of the whole coming from among the laborers, teamsters, builders and other similar occupations.

It is difficult, even impossible, to accurately interpret the statistics from which such proportions are derived because no allowance is made for the relative number of people in these two very general classes of occupation and because these figures are all derived from records of hospital to which laborers and out door workers are brought much more frequently than are those whose indoor work brings larger financial return. And yet while the figures 60-70 per cent are not accurate, still there seems no reason to question the general statement that people exposed by their work to the weather are more liable to pneumonia than indoor workers. This is probably the explanation for the greater number of cases of pneumonia in males.

Why exposure favors infection with the pneumococcus is not clear, but it has been suggested that direct exposure has an effect upon the ciliated epithelium of the bronchi, lessening their activity and thus favoring the penetration of the cocci to depths of the lungs not normally reached by them. Lipari has given experimental support to this idea by finding that animals given intratracheal injections of pneumonic sputum acquired pneumonia if they were just previously or just subsequently exposed to cold while they did not otherwise do so. The occurrence of pneumonia after ether or chloroform inhalations and without any aspiration of vomitus might be explained in the same way.

Dürck has for some years held the opinion that the lungs are not so free from bacterial contents as is generally supposed and bases his opinion upon the examination of the lungs of freshly slaughtered ani-

mals. He found in a considerable percentage of them that bacteria, both non-pathogenic and pathogenic were present. These bacteria appear harmless like the bacteria in the gastro-intestinal tract, until some favoring condition for their development arises. He found various forms of pus cocci and the pneumo-bacillus and pneumococcus. Of the influences which favor the development of bacteria he regards cold as particularly potent and was able to cause pneumonia in a considerable percentage of animals experimented on by exposing them to heat and then plunging them into cold water. His studies of the bacterial content of the lungs have been confirmed by Béco, who found the pneumococci twice in the lungs of 15 healthy house animals, by Boni, who found bacteria in 14 of 24 pigs, by Quensel who found them in the lungs of all of 16 calves, 5 horses, 6 pigs and in 11 of 15 sheep. W. Müller found the lungs sterile in but 5 of 22 rabbits.

The influence of cold has been studied by Lode, who, after shaving animals exposed them to cold and then injected culture of the pneumo-bacillus. 85% of animals thus treated died from pleurisy or pneumonia while only 12% of the animals injected but not exposed to cold died.

Clinical instances of pneumonia following an unusual exposure to cold are common and are practical examples of the conditions which Lipari and Dürck produced in the experiments cited in the preceding paragraphs, the only difference being that the pneumococci were already present in the upper air passages instead of being introduced at the time.

Every physician of experience has met enough instances of pneumonia following directly after exposure to a cold draft when overheated or after the clothing has been wet through to question the exciting influence of such exposure. Ziemssen in 205 pneumonias found 45 who referred their attack to exposure. Some allowance must be made for the fact that the general public, like the doctor of a few generations ago, are very prone to explain their ills on this basis and their reasons for thinking so must be inquired into each time. Ziemssen holds that in ten instances the proof seemed clear that the exposure did precipitate the attack. The time between the exposure and the onset of symptoms varied from one-half to sixteen hours. Grisolle determined the time between exposure and the onset of symptoms in 34 cases. In 18 of them general disturbances or local symptoms of the disease came on during the exposure or within a few moments, in 11 three hours intervened and in the others a period of one to two days elapsed before illness began.

Another curious but yet well established fact is the development of pneumonia shortly after trauma of the chest. It is not usual to include here the really rare cases of pneumonia following penetrating wounds of the thorax and the term traumatic pneumonia is applied only to the cases following contusions of the chest either with or without fracture of the ribs. The existence of such cases has been known for a long time but Litten was the first to give the matter systematic study and found that the cases were not so exceptional as might be supposed, for in 320 cases of pneumonia he found 14 instances

due to trauma, *i. e.*, 4.4 per cent. Demuth in 604 cases found 10 examples of traumatic pneumonia; Juergensen found one in 768 cases. Aufrecht says that he has found the traumatic cases no more frequently than Juergensen, *i. e.*, in 0.13 per cent. Stern who has made an exhaustive study of the influence of trauma in causing internal diseases says that the traumatic cases of pneumonia make up about *2.4* per cent. This is probably not far from the truth. The site of the pneumonia usually corresponds to the site of the injury, but may later extend to other parts. Thus Meunier found in a study of 25 cases of this sort that 18 traumas of the right side of the chest were followed 15 times by right sided pneumonia and 3 times by bilateral involvement, while 7 left sided injuries gave 7 left pneumonias.

Age and sex appear to have no influence except that trauma is commoner in the young and healthy adult male than in the other sex and at other ages. Alcoholism, however, is a contributory cause and an undue proportion of alcoholics are found in the traumatic cases. A previous attack of pneumonia is another important contributory cause.

In these as in other cases the active agent is the diplococcus pneumoniæ and the trauma in some way creates conditions favorable to the development of this bacterium. Because the essential cause is the same the clinical course, complications, outcome, etc., do not differ in these cases from those of any other group with this exception—hæmoptysis is more common and more severe than otherwise. The time elapsing between the trauma and the onset of the disease varies from a few hours to a day or two.

It seems natural to mention here the fact that pneumonia sometimes follows the inhalation of gases and fumes. In those cases accompanied by unconsciousness the pneumonia is usually the result of the aspiration of foreign bodies, food and the like. The occurrence of pneumonia following ether anæsthesia has already been mentioned and the suggestion made that in some of the cases at least it seems reasonable to assume a temporary paralysis of the cilia of the bronchial mucous membrane and consequent deeper penetration of the pneumococci accidentally present in the upper air passage or in the larger bronchi.

One of the most potent factors in increasing the liability to infection with the pneumococcus is a previous attack of pneumonia. The frequency with which this occurs is shown in the following table which also shows that the observations of different reporters varies greatly. The percentages range from the 9 per cent of Sears and Larrabee to the 40.3 per cent of Riesell. The truth is probably somewhere between the two.

Sears and Larrabee.	949	cases,	86	previous attacks
Stortz	280	"	73	" "
Morhart	133	"	55	" "
Pohlman	157	"	54	" "
Rychner	606	"	154	" "
Schapira	166	"	52	" "
Keller	140	"	44	" "
Grisolle	175	"	54	" "
Norris	500	"	57	" "
Pelletan	75	"	15	" "
Alison	80	"	23	" "
Riesell	253	"	102	" "

Ziemssen	93	cases	25	previous attacks
Griesinger	212	"	36	" "
Flindt	186	"	25	" "
Ebstein	438	"	81	" "
	4,443		936	

This gives a percentage of *21*.

The number of attacks which an individual may undergo is hardly limited. Chomel reports one patient who had pneumonia 10 times, Frank one with 11 attacks and Rush one who had 28 attacks. I have recently seen a child of eight suffering from what the mother insisted was the fifth attack of pneumonia. Of 397 patients who gave a history of previous attack, 140 had had more than two.

This unquestionable predisposition remaining after once going through an infection is the more surprising when it is contrasted with the rarity of relapse in pneumonia. Relapse occurs very rarely. Engel estimates that it happens in not more than one-half of one per cent of the cases. Wagner saw relapse but twice in 1,100 cases. Ruge had two relapses in 440 cases. It would seem as if the infection with the pneumococcus gave a transient immunity followed after an unknown period by a predisposition to reinfection.

CHAPTER IV.

GENERAL DESCRIPTION.

Of all the diseases which may be classified as typical, *i. e.*, those which generally follow so definite a course, going through certain changes from day to day with such constancy that they may be safely predicted and lasting a period so close to the average that the duration of the illness may be accurately estimated at its onset, of all such diseases there is none truer to its type than the pneumococcus pneumonia. And yet it is true, as Juergensen says: "There is no disease which presents so diverse a picture as the croupous pneumonia." Between the insidious, afebrile pneumonia of the senile and the stormy disease of the young child with severe meningeal symptoms, there is so very marked a contrast that at first glance they have no feature in common and yet in both instances the diagnosis must rest upon the same physical signs, and in neither instance can the diagnosis be made in any other way. The difference between the ordinary pneumonia and a primary pneumococcus meningitis, peritonitis or general sepsis is still greater and yet they are due to the same bacterium. While admitting freely the wide diversity in onset, course and outcome frequently seen in individual cases, I still believe there

is no truer typical disease than the croupous pneumonia and that even in the most widely diverse pictures there are certain features which suggest the existence of a common cause.

More often than not this disease comes on suddenly, frequently in the midst of sleep, with a severe, shaking, teeth-rattling chill. No matter what is done the patient feels cold. At the same time he becomes conscious of some difficulty in breathing and often complains of sharp cutting pain in the chest, oftenest in the region of one or the other nipple.

In other cases the onset is not so stormy. Instead of a single severe chill there are several slight chills or merely a chilly sensation. In children the onset may be with cerebral symptoms, convulsions or delirium. In still other cases the initial symptoms are nausea and vomiting. The temperature rises rapidly to 103-106 and with this the pulse goes parallel. The respirations are early increased out of proportion to the temperature and rapid pulse. A cough comes on, hard, dry and irritating, bringing up no sputum or only a little, tinged with blood and in some cases consisting entirely of blood. Even thus early it is sometimes possible to detect physical signs which enable one to locate the pneumonia. This may occasionally be done within twelve hours after the chill, but it must be admitted that this is rare. The first physical sign is an increase in resonance on percussion amounting in many cases to tympany. This hyperresonance lasts but a short time and is then replaced by the lessened resonance and dulness common in pneumonia. The temperature after once going up remains so

and is continuous in type. The pulse and respiration gradually rise. The urine shows the concentration common to febrile processes, but more commonly than in any other acute infection shows albumin and very frequently casts also.

The most characteristic symptoms, those upon which the diagnosis must always rest, are the physical signs discovered by examination of the chest. The respiratory movements are more rapid than normal and in place of the even, uniform, symmetrical movements, we find them jerky and asymmetrical, the affected side lagging behind and moving less completely than the normal side. In many cases the affected side is not only painful, but is tender to pressure and in some instances the area of superficial tenderness corresponds accurately with the diseased portion of the lung. The most important palpatory sign, however, is the increase in the vocal fremitus. This increase is usually conspicuous and is confined to the affected portion of the lungs. In some cases one can feel friction fremitus and the bronchial fremitus resulting from the secretion in the tubules. Percussion shows an area of dulness corresponding to the portion of the lung involved and inasmuch as this form of pulmonary infection usually affects an entire lobe or lobes the figure of dulness outlined corresponds very accurately to the surface projection of the lobe or lobes. The area should always be carefully outlined and the correspondence just mentioned is often a material aid in the diagnosis. The dulness is absolute, but there is not that deadness and sense of intense resistance experienced in percussing over fluid in the chest.

Auscultation shows three important physical signs: The fine crepitant râle, a sign of great importance, but not peculiar to pneumonia, as is sometimes stated; the bronchial breath sounds, and the bronchial voice sounds. In addition to these there is often friction.

Examination of the blood at this time shows with great constancy a polymorphonuclear leucocytosis and a properly made bacteriological examination of the blood will show the pneumococci. The sputum becomes somewhat more abundant, is very tough, stringy and hard to raise. It is often colored, the prune-juice color, and shows the presence of the diplococcus pneumoniæ, sometimes pure, but usually mixed with other organisms commonly found in sputum.

The disease runs on without much change from day to day, with the exception of more or less failure in strength until oftenest the sixth day, when suddenly the temperature falls, the pulse and respiration decrease in frequency almost to the normal, the patient breaks out into a perspiration. The whole picture changes in a few hours and the patient becomes convalescent.

This will serve as a brief description of the typical case. Unfortunately all too many of them do not run thus smoothly and the variation in the course will be discussed in the consideration of the individual symptoms.

CHAPTER V.

DESCRIPTION OF THE VARIOUS SYMPTOMS.

Mode of Onset. In a considerable majority of the cases the onset is sudden and marked by such severe symptoms that the patient can fix not only the day, but the hour and the minute of its beginning. Such patients will tell you that up to that moment they had been feeling perfectly well when like a bolt from a clear sky the chill came on. The proportion of cases beginning suddenly differs in different groups of cases, for example, approximately 75 per cent of pneumonias in young and healthy adults will begin suddenly, while among the aged and debilitated sudden, sharp onset is the exception and the disease begins so insidiously that it is impossible to determine the moment of onset. Sears and Larrabee in 949 cases report 660 as beginning suddenly, and of these 466 began with a chill, *i. e.*, 70 per cent began suddenly and 50 per cent with a chill. Norris records chills 250 times in 500 cases. Other authors place the percentage of cases beginning with a chill still higher. As before stated, the percentage is determined very largely by the character of the material studied.

In many cases the chill is absolutely the first symptom, but in others it is preceded by prodromal symptoms which are not severe and in no way sug-

gestive of a coming pneumonia. There may be malaise, anorexia, pain in the back, a moderate bronchitis, a mild sore throat, or other equally indefinite symptoms. Sears and Larrabee record such symptoms in about 20 per cent of the cases. Grisolle places their occurrence at a little higher level. Nausea and vomiting occur in about the same percentage of cases and diarrhœa somewhat less frequently. The gastro-intestinal prodromata are more common in children than in adults and when they or other prodromal symptoms are marked there is not apt to be any chill to mark the beginning of the active period of the disease. It seems likely that such prodromal symptoms are present much oftener than the statistical reports would lead one to think for in the presence of the severe symptoms characterizing the actual disease the patient will forget and the physician neglect to inquire for them. The duration of this prodromal stage varies from a few, two or three days, up to weeks, according to Traube, although this latter is uncertain and very exceptional if really so.

Besides these modes of onset, which are common, there are others which, while exceptional, are frequent enough to keep in mind. One of the most important is the insidious onset of pneumonia in old people. Without any chill, without cough, pain fever or acceleration of respiration, a pneumonia may develop in the aged and be discovered only accidentally or at the autopsy. This mode of onset is by no means rare in the old and in patients debilitated by any severe illness, and whenever such an individual appears to be feeling poorly or pre-

sents any variation from his normal average state, the lungs should be carefully examined for evidences of a pneumonia.

The sudden development of a delirium or a mania in an alcoholic should also suggest the same possibility, for it is not rare in such people to have the chill replaced by a delirium.

Convulsions mark the onset of pneumonia less often than in other acute infections. This is true also in children, except those who are very young and is the more striking when we recall the considerable number of other infections in children which begin in just this way.

Sometimes the first complaint of the patient is of abdominal pain, oftenest in the region of the gall-bladder or the appendix, and it is not uncommon to have patients with pneumonia sent to the hospital with a diagnosis of biliary colic or appendicitis.

Less frequently the onset is with multiple arthralgia with swelling of the joints, so that the case begins like an acute articular rheumatism. Nose-bleed and hæmoptysis mark the onset in a few cases. In still other cases the onset is more like that of a typhoid.

These insidious cases are always sources of anxiety, as may be inferred from the fact that they are often spoken of as the malignant or asthenic form of pneumonia.

The chill marks the point from which the course of the disease is reckoned. The duration and intensity of the chill vary very greatly. In some it is more a sense of chilliness and in these cases usually repeated, but in most the chill is a hard, teeth-rat-

tling chill, the patient feels terribly cold and no amount of covering or external heat brings any comfort. The skin is blue and cold. As a rule there is but a single chill, but it may be repeated. The chill lasts an hour or two, but may be continued for several. Chills, according to Aufrecht, are less common during the warm seasons than during the cold months, and less frequent in patients taken sick during the time they are in bed. At this time the body temperature is raised, although sometimes, as in a case reported by Bouchard, during the chill the temperature is actually subnormal. In this case the rectal temperature was 98.4° Fahrenheit during the chill. The temperature rises rapidly and within three to ten hours is at 102° to 105° F.

The modes of onset as here described are not those of a beginning pneumonia alone, but are common to infections with the pneumococcus wherever it may implant itself. Thus a pneumococcus tonsillitis, meningitis, general sepsis and the like are characterized by these same symptoms.

Another important truth is that pneumonias due to other bacteria are not apt to begin in the way characteristic of a pneumococcus pneumonia, and while it is too much to say that a typical onset means that the case is due to this bacterium, it is presumptive evidence. The significance of an atypical beginning is not so great for, as already stated, the pneumococcus pneumonia often varies from the type.

Temperature. The height to which the temperature rises varies greatly in different cases and differ-

ent groups of cases, ranging from no elevation at all to 108° to 110° Fahrenheit. Cases in which the temperature is normal or even subnormal are rare and occur only in the old or the greatly debilitated. Noica reports a case of pneumonia in a man of 64 lasting eight days, and the axillary temperatures ranged from 35.3° to 36.7° Centigrade (95.5—98° Fahrenheit) and the rectal temperature 36.4°—37.5° C. (97.5°—99.5° F.).

The accompanying table and chart, based upon 1,443 cases, will show the relative frequency of the recorded maximum temperature:

Under 100	1.8 per cent.
100—102	9.7 per cent.
102—103	20.9 per cent.
103—104	28.2 per cent.
104—105	26.1 per cent.
105—106	10.2 per cent.
106 and above	2.3 per cent.

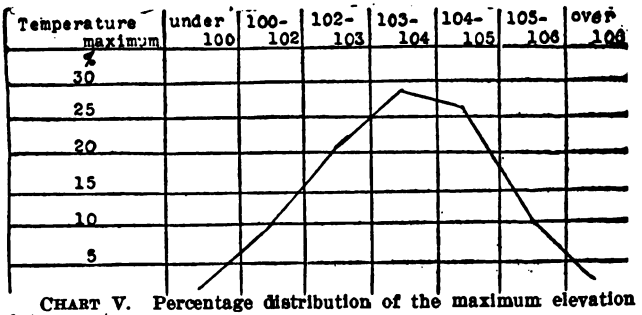


CHART V. Percentage distribution of the maximum elevation of temperature.

When once the elevation of temperature is established, it persists and assumes the type called

continuous, *i. e.*, the daily variations from morning to evening do not exceed a degree to a degree and a half Fahrenheit. There are, however, many exceptions in which the daily variation is greater than a degree and a half to three degrees, Fahrenheit, so that the temperature is of the remittent type. In some cases the temperature completely intermits, usually without any regularity in the intermission, but sometimes the fever may be as truly quotidian tertian, or quartan, as in malaria. Wunderlich recognizes three forms of intermittent fever in pneumonia, using the local condition in the lungs as the basis of classification. In one variety there is no change in the local signs to correspond with the intermissions and recurrence of the temperature; a second form, in which each intermission is accompanied by a like change in the local signs, and lastly, cases in which each new rise in temperature corresponds to an extension of the process into some new portion of the lung; the so-called wandering pneumonia. Not so sharply separated from these cases of intermittent course are the cases of relapsed pneumonia. As has already been stated, such relapses are rare, not more than one-half of one per cent., according to Engel, and in my experience far less often than this. The interval between the primary pneumonia and the relapse varies from one day to several weeks, and during the interval the patient is free from symptoms and the lungs present no, or only very insignificant, evidences of abnormal conditions. Japha, in a report upon six cases of relapsed pneumonia, put the interval between attacks at 1, 3, 5, 12, 16 and 26 days.

The relapse in five of these cases was in the site of the primary pneumonia. The temperature in the second attack rose sharply, either with or without a chill, and lasted a period averaging for the six cases five and a half days. Ruge and Grisolle say that generally the relapse is shorter than the primary attack, averaging 4.8 days, but it may be as short as three, two or even one day. In a personal observation of a relapsed pneumonia, the onset was insidious and all of the signs of consolidation were present before the patient made any complaint other than the indefinite one of not feeling well. Two weeks had elapsed since the first pneumonia had ended by lysis, and the temperature of the second attack lasted but two days.

Another variation in the type of fever is the irregular fever, exactly similar to that of sepsis. The temperature may remain continuously high for several days, but any moment may remit or intermit, and such remissions or intermission may occur several times a day or only at longer intervals. With such a temperature course there may or may not be repeated chills.

After continuing for from five to eight days, the temperature in cases which are following the type, falls by crisis, returning to normal or sometimes falling even below normal in the course of a few hours. The temperature may fall even as much as one to three degrees below the normal and remain so for two or three days. Such subnormal temperatures are not uncommon after crisis, but are exceptional in the cases in which the temperature falls by lysis. What shall be regarded as

a crisis is somewhat a matter of opinion, but generally a return to normal within 36 hours after the temperature begins to fall is regarded as a crisis, although some would regard it more as a short lysis. Juergensen states that he has seen a complete return of the temperature to normal take place in so short a period as four hours. Six to twelve to sixteen hours is commoner. Lebert in 110 cases gives the duration of the crisis as follows:

12 hours.....45 cases
24 hours.....25 cases
36 hours.....40 cases

The temperature may continue its even course until it ends by crisis, but in some cases there is a fall to normal, followed shortly by a rise to, or nearly to the point formerly attained. Such a fall is spoken of as a pseudo-crisis and occurs in about 10 per cent of the cases, 54 times in 500 cases reported by Norris. In still other cases the crisis is preceded by a short period during which the temperature rises and all the other symptoms become worse, a preturbatio critica.

The percentage of cases ending by crisis varies with the character of the material studied. In a general way crisis is commoner in children than in adults and in adults than in the senile. Cases which begin suddenly are apt to end by crisis. The following table shows the percentage in unselected material:

Aufrecht	1,501 cases,	873 ended by crisis
Croce	461 cases,	286 ended by crisis
Smith	60 cases,	27 ended by crisis
Sears & Larrabee	949 cases,	424 ended by crisis

Norris	500 cases,	301 ended by crisis
Townsend & Coolidge	212 cases,	128 ended by crisis
	3,683	2,039

This gives a percentage of 55.

It has been asserted by some authors, notably by Traube, that the crisis comes always upon an odd day after the onset of the disease, the 5th, 7th, 9th or 11th day, but it is easily proven that while the crisis is more often on an odd than on an even day it is far from being always so. Thus in 2583 ending by crisis as reported by Ziemssen, Wunderlich, Liebermeister, Aufrecht, Smith, Sears and Larrabee, Townsend and Coolidge 1479 *i. e.*, 57 per cent ended on the uneven days and 1104 upon the even days. Crisis may come on any day from the first to the twenty-second or later, but the greatest number come within a very few days. Thus in Aufrecht's table covering 873 cases ending by crisis no less than 45 per cent ended on the fifth, sixth and seventh day of illness and 70 per cent from the fifth to the ninth day.

The crisis may come on at any time in the twenty-four hours but in the majority of cases comes in the late evening or early morning hours.

In a not inconsiderable proportion of the cases the temperature falls by lysis as is shown by the following:

Sears and Larrabee.....	949 cases—	141 lysis
Norris	500 cases—	74 lysis
Townsend and Coolidge.....	212 cases—	84 lysis

Croce	461 cases—	175 lysis
	2,122	474

This shows a lysis in 22 per cent of the cases. Any case which presents a long continued lysis, say any lasting longer than twelve days, should be carefully and repeatedly examined to make certain in the first place that the infection is not due to some other organism than the pneumococcus, and in the second place that no complication has developed.

Pulse and Heart. At the same time with the rise in temperature comes an increase in the pulse rate which, generally speaking, is proportionate to the elevation of temperature. One must here as elsewhere take into consideration the individual and expect to find that in children the rapidity of the pulse is proportionately greater than in adults. It is also faster in women than in men and in small persons than in large. In adults the highest pulse rate in most cases ranges between 120 and 140 but there are many factors which will change these figures. The following table and chart based upon 1,064 adult cases will give some idea of the usual experience:

Below 100 maximum pulse rate.....	5.3%
100—110 maximum pulse rate.....	11.5%
110—120 maximum pulse rate.....	13.3%
120—130 maximum pulse rate.....	27.0%
130—140 maximum pulse rate.....	13.5%
140—150 maximum pulse rate.....	11.7%
150 and above maximum pulse rate..	7.8%

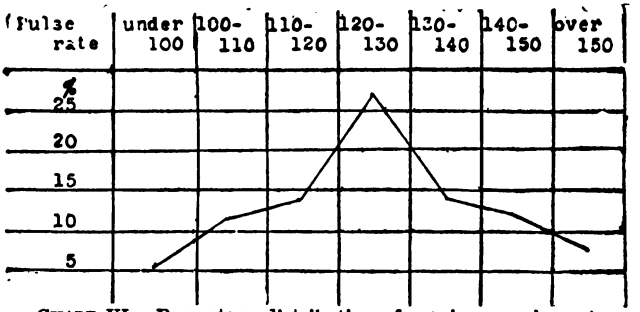


CHART VI. Percentage distribution of maximum pulse rate.

In a general way the pulse pursues a path parallel with the temperature, becoming more or less rapid as the latter rises and falls and when the crisis sets in the pulse falls rapidly to and very often far below the normal points so that a post-critical pulse rate of 60, 50 or even 40 is by no means exceptional.

The pulse rate has long been regarded as a factor in prognosis and justly so although it is easy to lay too much stress upon the point.

The mortality increases rapidly as the maximum pulse rate passes 130. It is hardly necessary to state that this applies only to healthy adults. In children even 150 or 200 may be reached without especial danger.

The relation of pulse to respiration in pneumonia is peculiar and is sometimes of value in suggesting the diagnosis early, before definite signs, which permit of a positive diagnosis, have developed. Normally this relation is 1 :4.5 but with the onset of a pneumonia the rate of respiration increases more rapidly than does the pulse so that the relation is

altered to 1:3, 1:2, or even in some cases 1:1. It is hardly necessary to say that there are many conditions other than pneumonia in which a similar change is observed but in an illness beginning suddenly with a chill, in absence of physical signs such a relation would strongly suggest the possibility of a beginning pneumonia.

During the earlier days of the disease the pulse is full, strong, of good tension and regular. It may continue so throughout the entire course but frequently the later days show a weaker and more rapid pulse something which need not however cause anxiety unless the weakness or the rapidity becomes extreme. An irregular pulse is much more disturbing and is the more so, the earlier the irregularity appears for it indicates either some previous disease of the heart muscle or more serious myocardial changes than usually result from the pneumotoxines. Irregularity of the pulse after the crisis is less dangerous but even then is serious enough.

Dicrotism of the pulse is common and of no essential significance.

Exact data as to blood pressure during the course of pneumonia do not, so far as I know, exist.

Examination of the heart itself in many cases shows no variation from the normal but frequently it is found enlarged to the right, the right border of the absolute cardiac dulness lying at or even to the right of the right border of the sternum. Such an increase is the result partly of overfilling of the right ventricle and partly of the yielding of its walls to pressure because of weakness of the musculature and yet providing the increase is not extreme one

should not be greatly alarmed by it unless there are other evidences of cardiac failure. The reason for this restriction upon the significance of the increase in heart dulness to the right is found in the fact that this increase may be due to an abnormal exposure of the heart, resulting from retraction of the lungs. Juergensen believes that because of the superficial breathing the lungs are less filled than normally and that the elastic tissue of the lungs, working against the lessened pressure of the air, causes the lungs to retract and thus exposes the heart to a degree which is not normal. Another factor present in cases of left-sided pneumonia is a displacement of the heart by the infiltrated lung. If in addition to the pneumonia, there is considerable exudate in the pleural cavity the cardiac displacement is still further favored. It is scarcely necessary to state that when a pneumonia involves the right middle lobe, the right border of the heart cannot be determined by percussion. Effort may be made in these cases to determine it by ausculatory percussion, a method which in my opinion has no value.

In estimating, in any given case of enlargement of the heart dulness, the relative importance of these three factors, displacement, uncovering and actual enlargement, one should pay attention to the intensity of the second pulmonary tone. So long as this tone remains accentuated, dangerous distention of the right ventricle need not be feared.

Because of the obstruction to the flow of blood through the pulmonary artery, always resulting from a pneumonia, the blood pressure in this vessel

is raised and the second pulmonary tone is accentuated and remains so as long as the right heart continues to do its work well. When it fails, the tone decreases in intensity, and such decrease may be the first clinical evidence of threatening heart failure.

The significance of an accentuation of the second pulmonary tone is less in cases of pneumonia involving the left upper lobe because the infiltration of that portion of the lung overlapping the pulmonary artery favors the transmission of the second tone and the tone may thus appear loud even though in reality it is less so than normally.

In pneumonia, as in all febrile diseases, systolic cardiac murmurs are not uncommonly heard and fortunately are rarely of any significance unless there is reason for referring the murmur to an antecedent valvular lesion. They should, however, always receive careful and repeated attention because of the possibility of an endocarditis developing as a complication of the pneumonia.

Another auscultatory phenomenon to which Fraentzel has drawn particular attention, is the gallop rhythm. This consists of three tones heard equally over the entire heart area, one tone systolic and two diastolic. It appears at the same time with dangerous cardiac weakness and has therefore a serious prognostic significance. Fraentzel has observed it at all periods in the course of pneumonia. It is of especially serious significance when it appears during the height of the attack. He observed it four times with other alarming symptoms just before the crisis, seven times on the day after the crisis and

fourteen times it was the first or a prominent symptom of collapse, thirteen times a fatal collapse, occurring at the height of the pneumonia.

Collapse from cardiac failure may occur at any time in the course of pneumonia. At the beginning or during the course, the body temperature remains high, although the surface temperature may fall, the patient becomes restless, often delirious, looks very ill indeed, the pulse becomes rapid, weak, thready and irregular. Respiration becomes difficult and the patient becomes cyanotic and is often more or less covered with cold perspiration. Such collapse as this is very generally fatal. Much less serious is the collapse which sometimes just precedes or accompanies the fall of temperature of the crisis. Collapse may also occur a few days after the crisis. Here also it is a very serious affair. In all of these cases the collapse is due to cardiac weakness, although in most instances the autopsy does not show any gross changes in the heart.

Lungs. The changes in the lungs and the physical signs and symptoms resulting from them are naturally the most important in the course of a pneumonia. Upon them the diagnosis must rest. It is impossible to insist too strongly upon the importance of the physical signs in the diagnosis of this disease. Even the layman is sufficiently familiar with the peculiar mode of onset and the symptoms of a pneumonia to suspect its existence, but no practitioner, no matter who or what he may be, should be willing to make a diagnosis of pneumonia without a careful and repeated examination of the thorax and its viscera. It seems, and should be,

superfluous to make such a statement in this day and age, but unfortunately there are still too many practitioners who neglect to examine the thorax and are content to make a diagnosis of pneumonia as a layman would. There would be less nonsense talked about aborting pneumonia if methods of physical examination were universally employed.

The three subjective symptoms referable to the respiratory organs of which the patient complains particularly are pain, dyspnœa and cough. The relative intensity of these vary in different cases and any or all of them may be absent.

Pain is one of the earliest and most constant of the symptoms, present according to Grisolle in about 90 per cent of the cases. In a very large majority of the cases the pain comes on within the first twelve hours, often at the same time with the chill and sometimes even preceding it. In some cases the pain does not come on early and we must then infer that at first the pneumonia was central and later extended to the surface of the lungs and to the pleura. The commonest site for the pain is the region of the nipple of the affected side, but it may be referred to the base of the lungs, to the back, the axilla, or in some cases the pain may be referred to some point remote from the thorax, oftenest to the abdomen. The anatomical explanation of abdominal pain is that the lower six intercostal nerves supply the abdominal wall as well as the parietal and diaphragmatic pleura, and when these nerves are irritated in their course the pain may be referred to their ultimate distribution, i. e., to the abdomen. Particular note should be made of the eleventh in-

tercostal nerve, which is distributed to the iliac region. Irritation of this nerve with referred pain has more than once caused suspicion of an appendicitis. Other nerves would cause pain referred to the umbilical, epigastric or hypochondriac region and excite suspicions of ulcer of the stomach, gall-stones or other disease of the gall-bladder. Pain may also be referred to the shoulder.

Pain is commoner in pneumonia of the lower lobes, the upper lobe pneumonias usually being free from pain. The reason for this is obvious. The pain is not due to the inflammation of the lung but to the pleurisy which is almost always present. It is due to the rubbing of one inflamed surface over the other and is in a general way directly proportionate to the amount of motion. There is little motion between the pleural surfaces of the upper part of the thorax, while there is a great deal in the lower part, hence the frequency and severity of the pain in this part of the chest.

The pain is not equally severe in all cases, even when the friction as heard by auscultation seems equally marked. The pain usually lasts only a short time but may continue for days. It is increased by deep breathing, by coughing and by pressure over the affected portion of the thorax.

Dyspnœa is one of the early and most distressing symptoms, indeed it may appear before any adequate explanation can be obtained by examination of the lungs. The severity of this symptom varies greatly. It is due to a variety of factors, especially the elevation of the temperature, the pain and the lessened breathing surface due to the infiltration

of the lungs and in some cases cardiac failure becomes an important element. The first of these is not of great importance for the acceleration of respiration due to this factor alone is not sufficient to greatly disturb the patient. The pain is, however, an element of importance. Because it is increased by deep breathing, the patient instinctively breathes superficially and endeavors by breathing rapidly to make up for the shallowness of each respiration. This factor is important in the causation of the early dyspnœa which characterizes some cases. Years ago Grisolle attempted to prove that pain had no influence upon the respiratory rate by comparing two groups of cases, similar in other respects but differing in that one group suffered pain and the other not. He found that there was no essential difference in the respiration rate. Traube objected to this and cited numerous instances of rapid breathing seen in painful affections of the abdomen without there being any other cause for dyspnœa. I think there can be no reasonable doubt of the fact that the pain on breathing does make the respirations rapid. The third factor in causing dyspnœa is the infiltration of the lungs. This lessens the breathing surface and nature attempts by breathing more rapidly to get more out of the remaining surface. Were this the only factor the dyspnœa would be proportionate to the amount of lung tissue involved, but the conditions are not so simple, and while in a general way this proportionate relation is true there are many exceptions.

Dyspnœa may also result from cardiac insufficiency and it makes an important part of the pic-

ture of collapse to which reference has already been made. Severe dyspnœa is a distressing and alarming symptom always, but it is especially alarming when neither the pain nor the extent of lung involvement is a sufficient explanation. Under these circumstances the possibility of its being due to failure of the right heart should occur to one even though there may be no other reason for thinking of it.

Even when the patient makes no complaint of dyspnœa the respirations will be found to be more frequent than normal. The rapidity is due as is

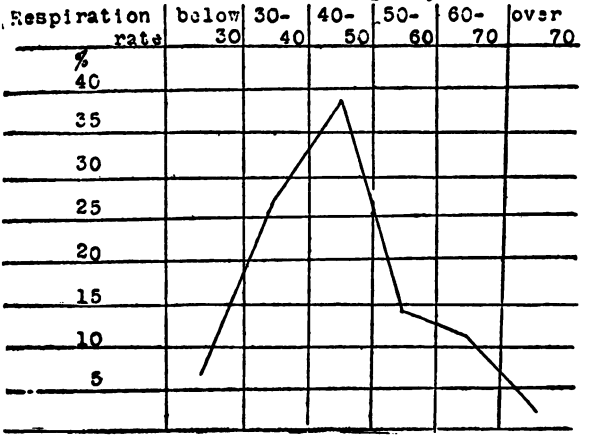


CHART VII. Percentage distribution of maximum respiration rate.

already indicated to a number of factors. To those already mentioned may be added the age or more particularly the size of the individual. The respirations are more rapid in a small person or a child than in another, other things being equal. In the

normal adult they increase to 30, to 40 or even to 50 or 60, although such increase as the last mentioned is rare and is almost always fatal. In children the respiratory rate is much greater, 60 to 80 being not uncommon. The following table and chart covering 740 cases over fifteen years of age as reported by Sears and Larrabee gives a definite idea of the frequency of respiration in pneumonia in general, although it will not serve as a guide in any special case:

Below 30	45	6.2%
30-40	200	27.0%
40-50	288	39.0%
50-60	107	14.4%
60-70	82	11.0%
70 and over	18	2.4%

With the onset of the crisis the respirations become slower but do not decrease so rapidly as do the temperature and pulse. The latter fall by crisis, but the respirations fall by lysis.

Cough is the third and sometimes the most troublesome of the subjective symptoms. It begins early, often appearing simultaneously with the chill and pain. It may be almost continuous, so much so as to seriously interfere with sleep or may be paroxysmal, each severe paroxysm being followed by a period of freedom and relative comfort. The cough is the more troublesome at first because it is practically non-productive. It is rarely if ever lacking entirely when the patient is a healthy adult, although it is less severe when the pneumonia is confined to the upper lobes. In young children, in old people, in the delirious and in those

whose pneumonia develops in the course of some protracted and exhausting illness there may be no cough throughout the entire course of the disease. The sputum is scanty, clear and tenacious. Very soon, however, it takes on those characteristics which are almost peculiar to the sputum of pneumonia. It becomes the rusty colored sputum. It is very stringy and so tenacious that frequently the patient cannot spit it out but must wipe it out of the mouth. It clings to the sides and bottom of the sputum cup and does not run out even when the cup is inverted. In exceptional cases the sputum contains considerable blood, enough indeed to warrant speaking of an hæmoptysis. Points and streaks of blood are not uncommon. The time of the appearance of the rusty color in the sputum has been studied by Grisolle, who gives the following table based upon 131 observations:

On the 1st day	45
On the 2nd day	31
On the 3rd day	14
On the 4th day	14
On the 5th day	11
On the 6th day	6
On the 7th day	5
On the 8th day	2
On the 11th day	2
On the 12th day	1

That is, the characteristic sputum appears within the first two days in 60 per cent of the cases and within the first four days in 80 per cent.

The amount of the sputum varies in different cases and in different stages of the disease, but is

FIG. 1. Cast of bronchus expectorated on the sixth day.

always small in the cases free from complications. Grisolle places the amount at 64 grams in 24 hours, Eichorst puts it at from 30-200 cubic centimeters.

Microscopically the sputum contains alveolar epithelium, red and white blood corpuscles in varying numbers and small masses of fibrin which on being spread out are found to branch dichotomously. These are casts of the smallest bronchi. Casts from larger bronchi are sometimes found. The bacteriological examination of the sputum is most important and should never be omitted, for upon the bacteria found much of the diagnosis and prognosis depends. If the diplococcus of pneumonia is found alone or nearly so the probabilities are that the case will pursue the average course, lasting the usual number of days and ending typically with complete resolution. If the sputum shows other bacteria to be present in excess or alone the prognosis becomes less favorable. For example, a pneumonia due to the influenza bacillus will last longer but no one can say how long and a very considerable number of them do not end by resolution but are followed by gangrene, suppuration or organization of the exudate. If the pus cocci are found alone or in large numbers, irregularity in the course is to be expected, the recovery will not be so smooth and the danger of abscess and gangrene is materially increased. The prospects are still worse if the tubercle bacilli are found either alone or in association with the pneumonia.

The demonstration of the diplococcus in the sput-

um is very simple and easy, requiring neither special skill nor special staining methods. It would be too much to say that the sputum in all cases of pneumonia should be examined for tubercle bacilli but certainly that of all cases presenting any irregularity in course or duration should be.

As the days go by the character of the sputum changes. It becomes more of a citron yellow color, is less tenacious and takes on more of the character of the ordinary muco-purulent sputum. It is expectorated more freely and more abundantly, but never reaches the amount one would expect from the amount of the exudate in the lungs.

There are certain complications which alter the character of the sputum. Pulmonary œdema, a common pre-agonal phenomenon, converts the sputum into the abundant thin frothy sputum characteristic of this condition. The onset of a gangrene brings about a complete change in the sputum. It becomes stinking, abundant, dirty brown, red, or green in color, and settles into the three layers com mon to the sputum of pulmonary gangrene, no matter what its cause. In case an abscess forms in the lungs or an empyema ruptures into the lung, the sputum becomes purulent, almost pure pus, and in many cases is raised in large amounts at intervals, while at other times there is little or no sputum.

The same conditions mentioned earlier as explanatory of the cases of pneumonia without cough, cause also pneumonia, without expectoration. Children, even those who have a cough, do not expectorate until they are several years old. The delirious, the insane, the debilitated, the aged may

run the course of a pneumonia without ever raising any sputum.

Important as the respiratory symptoms so far mentioned are, they cannot serve as the basis of a diagnosis either of the existence or of the location of a pneumonia. They are strongly suggestive but they are not diagnostic. The diagnosis of pneumonia must rest upon physical signs, and without these signs, a positive diagnosis cannot be made. The examination of the chest should be systematic and should be repeated daily or oftener in order that the physician may be informed as to the progress of the disease and warned of the development of complications.

Inspection and mensuration of the chest do not furnish much information. It is readily seen that the respiratory movements are more rapid than normal, that they are superficial and irregular, particularly so if the patient is suffering much pain. The affected side will also be found to be moving less than the normal side and to lag behind. This irregularity and asymmetry of motion is manifest only when the pneumonia affects the lower lobe. Measurement of the chest in these same cases may show a difference of circumference of ½-2½ centimeters, so little that it is of no value as an aid to diagnosis and for this reason measurement of the chest is rarely practiced and is quite useless.

Percussion is of the very greatest value in determining the existence and the site of a pneumonia. Under favorable circumstances the percussion note is altered even as early as twelve hours after the

onset of symptoms, although it must be admitted that demonstrable changes thus early are exceptional. The first change is an increase in the resonance often amounting to actual tympany, which is not influenced by opening and closing the mouth. Because of the beginning inflammatory process the lung tissue is relaxed, the alveolar walls are no longer tense and the air in the affected area vibrates as a whole and produces a tympanitic note on percussion. The lung tissue is relaxed as it is by moderate compression and the same tympany results. The physical explanation of this tympany is as Eichorst says not very satisfactory but there is no question of the fact that during the first stage of pneumonia as during the last a tympanitic note is often obtained on percussion.

With the infiltration of the alveoli, i. e., with the onset of the stage of red hepatization the percussion note becomes dull. The dulness, however, is rarely the intense dulness produced by an exudate in the pleural cavity, for the lung still contains air. When the upper lobes are involved, there may be tympany even at this stage, partly, according to Aufrecht, because in these cases the filling of the alveoli is never complete but also and more particularly because the consolidated lung tissue conducts almost without reduction, the vibrations set up by the percussion stroke to the air in the large bronchi and trachea and a tympanitic note results. This tympany is usually present only anteriorly and is limited to the first and second intercostal spaces.

This is the so-called Williams tracheal tone and the pitch of this tone is altered by opening and closing the mouth. Sometimes, but certainly rarely, a similar tympanitic element in the percussion note is present over lower lobe pneumonias. Bäumler and Juergensen hold that this is due to vibrations set up in the columns of air present in the bronchi of the affected lobe. Tympany over a consolidated lower lobe may also be due to the vibration of air in the stomach or colon.

One very important peculiarity of the dulness elicited by percussion over a pneumonic lung is that there is not the absolute deadness and resistance experienced on percussing over an organ containing no air or an exudate into the pleural cavity. There is the same difference in the "feel" on percussing a pneumonia and a pleurisy with effusion as there is in percussion of a lath and stringer partition and one made of brick. This difference is difficult to describe, but is one which is readily appreciated and one which under circumstances is a valuable aid in diagnosis. If the afferent bronchus of the affected area of the lung is plugged by exudate, the dulness is absolute and is like that of a pleurisy.

The strength of the percussion stroke should not be excessive and in cases which are not perfectly clear both light and heavy percussion should be practiced. This precaution is less necessary in the lobar than in the lobular pneumonia, but when the process begins centrally, as it is may do, light per-

cussion might reveal nothing, while heavy or deep percussion would show an area of dulness.

The area of dulness corresponds in site, shape and approximately in size to one or more of the lobes of the lungs. The size of the area, as stated earlier, is somewhat larger than that described as normal. The line of division between the upper and lower lobes starts opposite the second or third dorsal spine and passes downward and outward to end over the sixth rib in the neighborhood of the nipple line. This fissure is practically the same on both sides and marks the upper and anterior border of the lower lobes. On the right side the fissure divides about 2 to 2.5 inches above the angle of the scapula. The lower division has just been described; the upper division passes almost horizontally forward to meet the anterior border of the lung at the level of the fourth or fifth ribs. Between the fissures is the middle lobe and above the upper one lies the upper lobe.

The croupous pneumonia, if once started in a lobe, almost always involves the entire lobe and therefore the percussion figures correspond accurately to the areas indicated above. There sometimes arise conditions in which it is difficult to say whether an area of dulness is due to a pneumonia or to some other condition. If the dulness corresponds in shape, site and size to one or more lobes the process is almost certainly a pneumonia. For example the physical signs of a pleurisy with effusion are exactly the same as those of a pneumonia with plug-

ging of the bronchi and the other symptoms of these two affections are not sufficiently different to enable one to distinguish them with certainty. Under such circumstances, and they are not so exceptional, an area of dulness shaped like that of the lobe in question is sufficient to warrant the diagnosis of a pneumonia rather than a pleurisy.

The relative frequency with which the various lobes become the site of a pneumonia has been studied by various authors and the conclusion, based partly on clinical and partly on pathological data, is as follows:

In 14,914 cases collected by different reporters, the distribution to the right and left lung is as follows:

Right lung....7,900 cases, i. e., 53 per cent.
Left lung.... 5,236 cases, i. e., 35 per cent.
Both lungs....1,778 cases, i. e., 12 per cent.

These figures must be very nearly correct for they correspond almost exactly with those given by others.

Aufrecht gives the following: right 52%, left 35.2%, both 12.8%, and Huss gives right 53%, left 32% and both 15%. To put it in another way, the right lung is involved in 65 per cent and the left in 47 per cent of all cases of pneumonia.

Ziemssen long ago noted that these figures did not hold for children, for in 191 cases he found the right lung alone involved in 47.7 per cent, the left only in 46.0 per cent, and both lungs in 6 per cent. Aufrecht suggests that this difference between

the distribution of pneumonia in children and adults is due to the greater use of the right than the left arm in adults, but this seems scarcely better than no explanation at all.

When one compares the frequency with which the upper, lower and middle lobes are involved, irrespective of whether they are right or left, the lower lobes are found to be affected much more frequently than the upper. Thus, in 9,563 cases, involving 11,321 lobes, the

Upper lobes were affected. 3,127 times, i. e., 27.6%
Lower lobes were affected. 6,658 times, i. e., 58.8%
Middle lobe was affected... 1,536 times, i. e., 13.5%

The lower lobes are involved about twice as often as the upper, and the upper about twice as often as the middle. Still further detailed, the following distribution is found in 9,333:

Right U. L.....	1,129	12.0	per cent.
M. L.....	188	2.0	per cent.
L. L.....	2,316	24.8	per cent.
Right two lobes..	671	7.2	per cent.
Right lung.....	907	9.7	per cent.
Left U.....	583	6.2	per cent.
Left L.....	2,242	24.0	per cent.
Left lung.....	751	8.0	per cent.
R. & L. U.....	91	0.97	per cent.
R. & L. L.....	368	3.8	per cent.
R. U. & L. L....	53	0.57	per cent.
R. L. & L. U....	34	0.36	per cent.
	9,333		

This does not exhaust the list of possible combinations, but others are so infrequent that they may be neglected.

The dulness over the affected area continues through the stages of red and grey hepatization, so long as the alveoli are filled with exudate. When this begins to undergo absorption and air again enters the alveoli, the dulness is replaced by hyperresonance or tympany which in this stage are more distinct and more frequently found than in the first period of the disease. When the lung has returned to its normal condition, the percussion note is again normal. As already stated, the time required for this varies from as short a period as one day up to several weeks.

Something should be said of the influence of the pleural exudate present in most cases of pneumonia. Usually it is small in amount, fibrinous and located over the affected lobe or lobes. In such cases it has no influence upon the physical signs either of percussion or palpation, but when considerable in amount it has much effect. The dulness of the pneumonia is increased to the more intense and the more resistant dulness of a pleural exudate. This effect is, however, insignificant when compared with the changes an abundant pleural exudate makes in the palpatory and ausculatory signs of pneumonia.

Percussion of those portions of the lungs which border upon a pneumonic area shows some changes, but unless there is a considerable accumulation of

exudate in the pleural sac they are not marked. The percussion note is somewhat hyper-resonant, partly because of pressure and consequent relaxation, and partly because of a compensatory emphysema of the healthy parts of the lungs, resulting from an effort to assume the function of the pneumonic area.

Palpation of the thorax yields valuable aid in the diagnosis of a pneumonia. The vocal fremitus is increased over the infiltrated area. The vocal fremitus of the two sides should always be compared and palpation should be made not only with the flat of the hand but also with the ulnar edge. The latter method is of particular value in outlining the area of increased vocal fremitus. In favorable cases the infiltrated area can be outlined as accurately by this linear palpation as it can be by percussion.

There are a number of circumstances which alter the strength of the vocal fremitus so that it is usual to find that it changes from day to day and even from hour to hour. If the bronchi going to an infiltrated area are plugged the vocal fremitus of necessity disappears, there being no column of air along which the vibrations from the larynx can be transmitted. If the obstruction to the bronchus is of such a nature that it can be readily dislodged, an effort at coughing will cause the vocal fremitus to reappear. In some cases the bronchi are plugged by fibrin and the vocal fremitus may be entirely lacking for days at a time.

If the amount of exudate in the pleura is excessive the vocal fremitus is decreased often even to complete disappearance. If this is the cause of feeble or absent vocal fremitus, coughing will of course have no effect upon it and the fremitus can reappear only when the exudate is absorbed completely or at least reduced to a small amount.

The other phenomena elicited by palpation are not of great importance but may properly be mentioned. When the chest walls are thin the various râles determine the character of the fremitus. Even the fine crepitant râles may be felt.

The friction of the accompanying pleuritis may also be felt in some but by no means in all cases. Even in cases where the friction is marked as shown by auscultation there may be no friction fremitus.

Benczur and Jonas some years ago drew attention to a method of thermo-palpation. By passing the hand lightly over the exposed skin, differences in temperature in different zones could be made out. The skin over air-containing organs shows a higher temperature than that over solid organs; is warmer, for example, over the lungs than over the heart or liver. The surface over a pneumonic area is warmer than over the normal lung. This method of examination is not of great value and the reports by various experimenters have been contradictory.

Auscultation. The earliest and one of the most important signs of pneumonia is the crepitant râle. This râle is a very fine one, well compared by Williams to the sound produced by rolling the hair

between the fingers close to the ear. It is developed in the alveoli and the finest bronchi by the passage of the inspired air through the lumen of the bronchioles into the alveoli, the communicating opening being narrowed either by swelling or secretion. It occurs only during inspiration and ceases as soon as the alveoli are filled. The respiratory murmur can be heard at the same time. This râle is present in all cases examined during the congestive stage of the pneumonia, but its duration is often very short. It is frequently spoken of as pathognomonic of this disease, but this unfortunately is not true, for it occurs in any other condition in which fluid and air are both present in the alveoli. The other conditions in which this râle is heard are usually easily differentiated from pneumonia, so that the fact that the crepitant râle occurs elsewhere than in pneumonia is not so confusing as it otherwise would be. The crepitant râle is heard in pulmonary œdema and in hæmorrhagic infarcts of the lungs, both of which are easily distinguished from pneumonia. More important is the fact that febrile patients who lie without change of position upon the back often show very distinct crepitant râles over the lower and posterior parts of the lungs, especially when their usually superficial breathing is on order replaced by a deep inspiration. This fact should be kept in mind and one should not be misled into diagnosing a complicating pneumonia in cases of long continued confinement to bed with some febrile disease like typhoid merely upon the basis of crepi-

tant râles. This same râle is met with also in beginning catarrh of the finer bronchi in which there is no reason to think of a broncho-pneumonia.

When the pneumonia is centrally located one may hear only sharp, puerile breathing, or faint though still vesicular breathing with râles of various sizes larger than the crepitant râle. If the process extends to the periphery, as it usually does, the crepitant râle appears.

With the filling of the alveoli in the stage of red hepatization the crepitant râle disappears to reappear when the exudate is undergoing absorption and the air is again entering the alveoli. In some cases this râle can be heard throughout the course of the pneumonia, arising in those parts of the lung which border upon the consolidated area and have undergone the pathological changes which create the physical conditions necessary for its production.

In most cases other râles than the fine crepitant râle are heard arising from secretion in bronchi of larger calibre. They may continue throughout the course of the disease and even when not heard early, they appear with the softening of the exudate and the more abundant or more properly more fluid secretion of the stage of resolution.

As the disease advances the breath sounds become harsher and the expiration longer and by the time the alveoli are filled with exudate we find the breathing bronchial. This is a peculiar character of breath sounds for which we have in English no word which is so descriptive as the German "hau-

chend." The bronchial breath sound is loud, seems close to the ear and resembles in character the sound heard normally over the trachea, though it is less intense. It is heard during both inspiration and expiration. Another important and but little appreciated peculiarity is that it can be heard much more distinctly by direct auscultation than through the stethoscope. I have no explanation to offer for this, but there can be no question of the fact. This peculiarity is most important in cases of central pneumonia and in all cases which are seen early for direct auscultation will often enable one to hear bronchial breathing a considerable time before it can be made out with the stethoscope. The direct auscultation is of especial value in children, enabling one to detect foci of consolidation easily overlooked and in febrile cases where one is called upon to decide whether crepitant râles heard over the base of the lungs are due to a beginning pneumonia or merely to the fact that the patient has been lying too constantly upon the back.

The bronchial breathing continues throughout the consolidation stages of the pneumonia, providing only that the afferent bronchi are patent. It will sometimes happen that they are plugged by exudate. If this can be dislodged by coughing the bronchial breathing appears, otherwise it does not. Under such circumstances one may listen many times without hearing any bronchial breathing, but fortunately such cases are exceptional.

The bronchial breathing is in no way peculiar

to pneumonia, for it is the result of the physical condition of the lungs rather than of any special pathological process. Anything which forces the air from the alveoli while leaving the bronchi open will produce the condition necessary for bronchial breathing. The commonest and therefore the most important of these conditions is the compression of the lungs by fluid in the pleural cavity. Under these conditions the bronchial breathing is usually limited to the borders of dulness caused by the fluid, i. e., is heard over the compressed lung, but is not heard through the pleural exudate. Sometimes, however, the sound is transmitted perfectly through the fluid in the pleural sac and a combination of signs is produced very closely resembling those of pneumonia.

Another physical sign which results from the same conditions which produce the bronchial breathing, i. e., airless alveoli with patent bronchi, is the bronchophony or bronchial voice sounds. When auscultation is made over a pneumonic lung, while the patient is speaking, the vibrations from the vocal cords are transmitted so readily and perfectly through the solid lung that the voice sounds nearly as loud as when one listens to the voice direct. There is some reduction in intensity and the character of the tone is altered and the articulation is less pure. In some cases it is well to listen both to the normal and to the whispered voice. The change in character is so peculiar and so sharply confined to the infiltrated area that in favorable cases the pneumonic focus can be outlined as accurately as by percussion. That which was said in regard

to methods of auscultation of the bronchial breath sounds could be repeated with equal truth here. Direct auscultation is preferable to auscultation with the stethoscope. The bronchial voice sounds disappear with the softening of the exudate.

The pleurisy, which forms so constant a part of the pathology of this disease, frequently shows itself by friction, the intensity and duration of which varies greatly in different cases. If the amount of exudate is large it modifies the physical signs greatly, causing a reduction or absence of the crepitant râle, the bronchial breathing and the bronchophony.

Blood. While even the ancients knew that there were certain changes which to a degree distinguished the blood in pneumonia from the blood in other febrile diseases, no progress was made and no really practical information was gained until the introduction of modern methods of study of the blood. Within the last few years the study of the blood in pneumonia has shown facts which have altered our conception of the disease, have increased our ability to recognize it, to anticipate the development of complications, and, moreover, it has shown clearly, what was formerly only dimly suspected, that the pneumococcus causes other clinical pictures than the classical pneumonia.

The change which has been known the longest is the increase in the amount of fibrin which characterizes most cases. The increase is very considerable and is easily demonstrated. It is so constant in its occurrence that when it fails to appear the case must be regarded as atypical and therefore more serious

than it otherwise would be. This increased amount of fibrin has no particular bearing so far as is known upon the symptoms or complications of the disease. It is interesting to note in connection with this that phlebitis, which one might expect to be common in a disease characterized by an increased coagulability of the blood, is as a matter of fact a rare complication of pneumonia. Turk has drawn attention to the interesting fact that in the cases which show a leucopenia the fibrin is usually deficient.

Changes in the red blood corpuscles are constant, but not so important as those in the white blood cells. The frequent occurrence of jaundice and of hydrobilirubinuria are evidences that there is an often considerable destruction of the red cells and yet the count is usually high; there may be even a polycythæmia amounting in some cases to 7,000,000. This is probably due to a decrease in the total amount of blood, caused, as Bollinger suggests, by the massive exudate in the lungs. In some cases, however, there is an actual decrease of 500,000-1,000,000 red blood cells during the febrile period. Usually, however, there is not much secondary anæmia and such as occurs does so after the defervescence. The only exceptions to this rule are the cases in which complications such as an empyema, an abscess of the lung or the like develop. During convalescence, nucleated cells, either normoblasts or megaloblasts may be found.

The hæmoglobin decreases and as in all secondary anæmias falls more rapidly than does the number of red blood corpuscles. The percentage rarely falls below 65 or 70 except in the complicated cases.

The important changes are in the number of leucocytes. Excluding certain mild and some of the very severe cases, a leucocytosis is found in all. The degree, the date of the appearance and the duration of the leucocytosis is liable to many variations. As a rule it appears very soon after the chill, although it must be stated that circumstances which permit early blood counts are unusual. Ewing found a leucocytosis of 25,000 four hours after the initial chill; Rieder and Laehr found an increase six hours after the chill. In many cases the leucocytosis does not appear until later and as already stated fails to appear at all in some cases.

In a general way the leucocyte curve is parallel to the temperature curve and may therefore be expected to rise sharply when the temperature comes on suddenly and gradually when the onset of the temperature is gradual. The same parallelism is shown at the end of the disease, the leucocytosis ending by crisis or lysis as the temperature does. It should be kept in mind, however, that the conditions are not so simple that we may expect an absolute parallelism. There are many exceptions to the rule. For example, a case may show a well developed leucocytosis which continues for a few days and then without any amelioration of the symptoms disappears. Such cases are usually fatal. Or with the onset of the crisis the leucocytosis continues instead of coming to an end. Such cases will be found to have some complication.

The maximum leucocytosis is usually shortly before the crisis and after the crisis the count decreases rapidly. In case of a pseudo-crisis the leucocytes do

not decrease in number, a fact which will often enable one to distinguish a false from a true crisis.

The degree of leucocytosis varies over a wide range and appears to be directly proportional to the reaction of the organism to the infection. If the infection is mild, or if the reaction of the organism is slight, there is little or no leucocytosis. Insufficient reaction is the more frequent happening and cases without leucocytosis are to be greatly feared.

A leucocytosis of 6,000 to 12,000 is mild, one of 18,000 to 20,000 is moderate and from there up is severe. The highest leucocytosis known to me is that recorded by Laehr of 115,000. In general the leucocytosis is higher in children than in adults. Sears and Larrabee state that of 211 cases in which leucocyte counts had been made all but 29 showed over 10,000. Cabot gives more detailed reports of 186 cases with the following table:

Cases with leucocytosis of	10-15,000	24
	15-20,000	49
	20-25,000	46
	25-30,000	19
	30-35,000	4
	35-40-000	7
	45-50,000	2
	50-55,000	4

Practically 75 per cent of these cases showed a leucocyte count of from 10-30,000.

The factors which determine the degree of leucocytosis are not understood and for the present we can only say that if the organism reacts vigorously to the infection the leucocyte count is high. Ewing says that in 47 cases which reacted vigorously the

average leucocyte count was 30,000; in 27 with a moderate reaction, the average was 20,000; in 27 with deficient reaction, the average was 9,000, and in 12 asthenic cases there was a hypoleucocytosis.

Ewing also draws attention to the relation between the extent of the lung involvement and the degree of the leucocytosis. Thus:

63 cases with one lobe averaged..20,000 leucocytes
24 cases with two lobes averaged.22,700 leucocytes
12 cases with three lobes averaged 25,000 leucocytes
1 case with four lobes showed..27,000 leucocytes

I think it is easy to exaggerate the influence of the extent of pulmonary exudate, an opinion naturally suggested by the fact that cases of pneumococcus infection without any pulmonary involvement show a high leucocyte count. As an example may be cited a recent case of pneumococcus sepsis in which the leucocytes ran as high as 48,000, but there was at no time any pneumonia nor indeed any localization of the pneumococcus in any organ. The leucocytosis is not the result of the pulmonary changes, but is due to the pneumococcus, and we can say that if a severe infection occurs in a strong and healthy individual there will be a leucocytosis. And yet this very general statement, made in terms which cannot be accurately defined, must be modified to this degree. In some cases occurring in apparently healthy adults there is no leucocytosis and experience has taught us that these cases are generally rapidly fatal. The infection seems so severe that the organism is overpowered and can make no response, no effort to protect itself.

The type of white blood corpuscle which is in-

creased is the polymorphonuclear neutrophile leucocyte. These are increased so that they make up 80-95 per cent of the cells. Turk has counted as high as 96.5 per cent and counts over 90 per cent

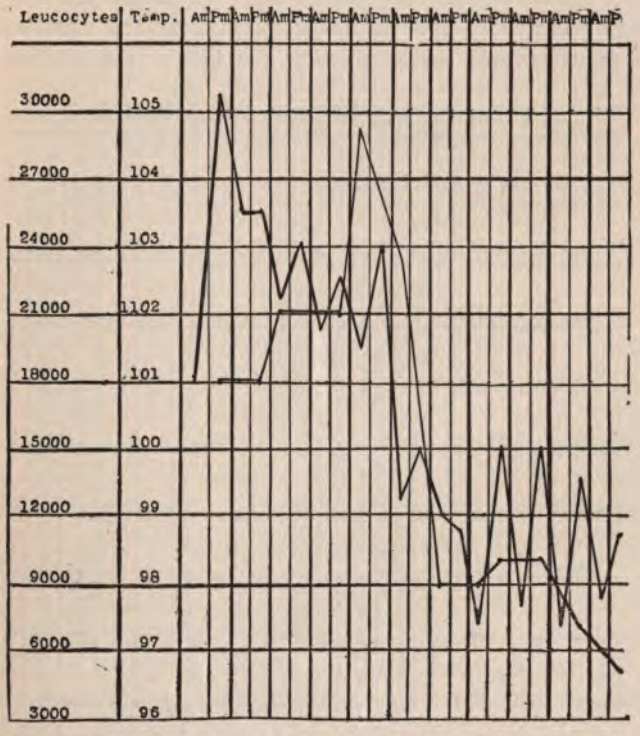


CHART VIII. Curve showing precritical leucocytosis. Heavy line, temperature; light line, leucocyte count. (From Limbeck.)

are not exceptional. The lymphocytes are reduced relatively and often absolutely, falling as low as 2-4 per cent. The large mononuclear type persists and may even be increased. The eosinophiles are

few and far between. These statements, however, are liable to exceptions, as is illustrated by the case which Cabot reports as showing 66 per cent of lymphocytes in 94,600 whites.

When the defervescence sets in the polymorphonuclear cells decrease rapidly in number, the lymphocytes increase in proportion, the large mononuclear

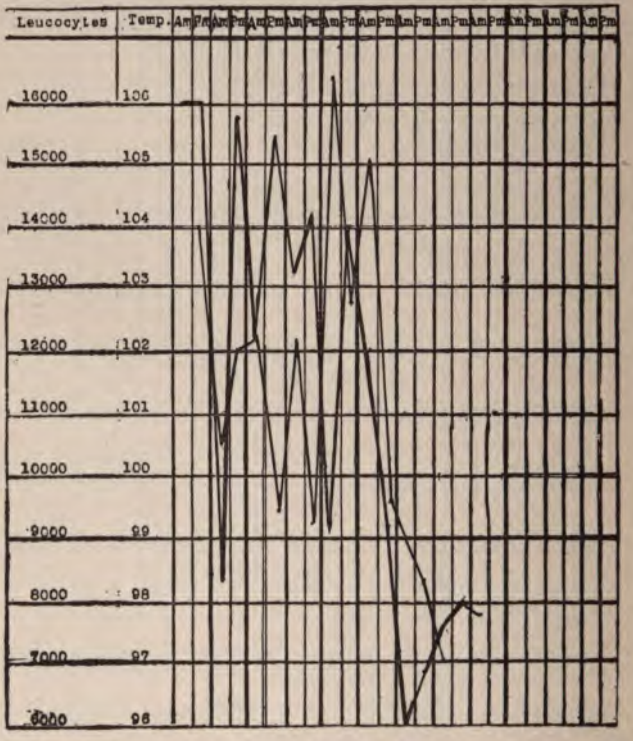


CHART IX. Curve showing comparative course of leucocytosis and temperature. Heavy line (starting at 106 and dropping to 98 2-5), temperature; light line (starting at 14,000 and dropping to 10,600), leucocyte count. (From Laehr.)

form may reach 16 per cent. Myelocytes occur in all cases during the defervescence when they may reach 10 per cent or more.

In some ways more important than the number of white blood cells is the bacterial content of the blood in this disease. It has been known for years that pneumococci occur in the circulating blood, but only recently has it become evident that their presence there is not exceptional, but usual. They have been found in so large a percentage of cases of pneumonia that we are justified in saying that the few failures are due either to insufficient search or to the fact that the case was not a pneumococcus pneumonia.

Rosenow, who has done more and better work upon this subject than any one else, reports but thirteen failures in 145 cases, i. e., in only 8.9 per cent, and in two of the failures the pneumococci were found in the smears although the cultures remained sterile. In most of the other cases the failure is sufficiently accounted for by the fact that the cultures were made late, after the temperature had become normal.

The bacteria are sought in two ways, by smears and by cultures. The former method is the easier, but is much less certain. Blood smears are prepared and fixed in the ordinary way and then stained with any of the aniline solutions or by Gram's method. In some cases the bacteria are so numerous that they are found in every field in every smear, but usually rather long and careful search is required to discern them.

The cultural method is much more certain. The

arm is carefully disinfected after being washed with green soap. A constrictor is then put about the arm well above the elbow, exactly in the manner for a venesection. This fills up the veins and makes their puncture easier. The syringe used to aspirate the blood must be built in such a way as to admit of easy and complete sterilization. The best for the purpose is an all-glass syringe. After sterilization of the syringe, needle and field of operation, the needle is plunged through the skin into the median basilic or median cephalic vein and five to ten cubic centimeters of blood are withdrawn. This is usually a very easy and simple procedure, becoming difficult only when the patient is so fat as to make it hard to find the veins and in children.

The culture medium which proves best for this purpose is ordinary bouillon. It must, however, be used in rather large amounts, best 50 to 100 c.c., and each tube should receive 1-2 per cent of the blood. Three or four cultures should always be made. This increases the chances of success and if one tube becomes contaminated the others are at hand for study. The cultures are grown at body temperature and the growth shows in from thirty-six hours to four or five days.

The opportunities for examination of the blood early in the disease are not common, but the bacteria have been found as early as twelve hours after the initial chill. In a general way the cultures are more easily obtained during the earlier days of the disease than just before or after the crisis. Sometimes, however, the pneumococci are found easily even days after the crisis. More frequently we

have occasion to make the culture before the diagnosis is made from physical findings. Thus Rosenow mentions four cases in which this occurred. It is, however, in cases which at no time show pulmonary changes that this method of examination has proved of the very greatest clinical value, throwing a flood of light upon cases which would otherwise remain obscure. I cannot express myself too forcibly upon this point, nor urge too strongly the employment of blood cultures in all cases of obscure infection.

Comparative studies of leucocytosis and pneumococcaemia do not as yet yield any definite results. The pneumococci are found in cases of high and of low leucocytosis, and may be missed with either. There seems also to be no relation between the number of bacteria and the degree of leucocytosis. They may be found in large numbers in spite of a leucocytosis, or may be sought many times in vain, although there is a leucopenia.

It was formerly thought that the presence of pneumococci in the blood was of unfavorable significance, but most believe, and this is my opinion, based on repeated observations, that they have no bearing upon the prognosis. While of the very highest diagnostic significance, they are of no consequence prognostically.

The agglutinating action of serum of patients suffering pneumococcus infection is not yet sufficiently studied to be of practical value.

Skin. This may seem an odd, as it is certainly an unusual place, to consider the skin changes in pneumonia, but it is placed here because of the be-

lief that it should be included among the more important of clinical manifestations of this disease. Herpetic eruptions are so common that their appearance in the course of a febrile process should at once suggest the possibility of a pneumococcus infection, no matter how much the clinical picture may differ from that ordinarily associated with this bacterium. Herpes is a common symptom in but three febrile diseases, malaria, the pneumococcus infections and epidemic meningitis. Which of these diseases should be first suggested by a herpes is determined largely by the place in which the patient lives. In malarial districts, malaria is the natural thought, but in all other portions, pneumonia, or more properly a pneumococcus infection should be the first thought. The frequency with which herpes occurs is variously stated and no doubt at different times occurs with different frequency. The figures are variously given as from 13 per cent to 50 per cent. Even the latter figure is in my opinion too low.

The herpes may appear at any time in the course of the disease, from before the chill to after the crisis, but it is usually seen first about the second or third day. The number of vesicles ranges from one or two to dozens, or even hundreds. A single vesicle is very common and this, I believe, explains the small percentages given by some authors. Unless one is fully impressed by the diagnostic importance of this trivial symptom, a single vesicle is easily overlooked, especially as they are not infrequently within the nostril or hidden in the hair of the moustache. The vesicle is usually superficial,

only rarely causing enough destruction of tissue to leave a scar.

Their commonest site is somewhere in the course of the middle branch of the fifth nerve, usually only one, but sometimes both sides being affected. The blisters are common upon the alae of the nose and the neighboring skin.

Less often they are found over the lower branch of the fifth, upon the chin or lips and only rarely are they seen over the upper branch.

They may, however, occur over the distribution of any nerve. I have thus seen them over the intercostals, over the last sacral and over the course of the lower cervicals and upper dorsals extending down the inner surface of both arms to the wrists.

Howard, in 1903, published an interesting study of the herpes of pneumonia based upon the study of two cases with autopsy. One of these showed a herpes zoster and the spinal ganglion of the corresponding cutaneous nerve showed definite changes consisting of congestion and hemorrhage, cellular infiltration and degeneration of the ganglion cells. The other case showed a labio-nasal herpes and the corresponding Gasserian ganglion showed changes similar to those described as occurring in the spinal ganglion.

Geissler believes that herpes has also a prognostic significance, of 182 cases with herpes 17, *i. e.*, 9.3 per cent died while of 239 cases without herpes 70, *i. e.*, 29.3 per cent died. Ten of fifty cases over thirty years of age, who showed herpes, *i. e.*, 20 per cent, died, while 53.2 per cent of 109 over thirty and without herpes died.

Sweating is common at any stage in the course of a pneumonia, but particularly at the time of crisis, when the perspiration may be so profuse as to soak the bed. Sudamina are common.

Other forms of eruption are rare in the course of this disease, but sometimes one may see hemorrhages into the skin. The disease may be associated with a hemorrhagic diathesis just as any other infection may, but is so only very infrequently. This is the more surprising when one recalls the frequency of cutaneous hemorrhages in the epidemic meningitis. This disease has so many clinical features in common with pneumonia and the resemblances between the pneumococcus and the diplococcus intracellularis are so numerous and so close, that I should not be surprised if it were finally proven that the two diseases are merely different effects from the same bacterium, in other words, that the pneumococcus and the meningococcus are one and the same. The labial herpes, as already stated, is very common in the course of epidemic meningitis, and it seems strange, in view of the facts just stated, that purpura should be so rare in pneumonia.

Brain and Nervous System. The intensity and character of the nervous symptoms appearing in the course of pneumonia vary greatly, but it is rare that one finds a case entirely free from them throughout the entire course. They range from a moderate headache with a slight nocturnal delirium at the height of the disease to the most violent delirium and mania, from slight stupor to the deepest coma, from convulsions to paralysis. The ordinary case complains

of some headache, is restless or a little stupid, usually a little flighty at night or even during the day, presents in short the ordinary nervous phenomena of an acute infection. Any one of these symptoms may, however, become the ruling one in the clinical picture. For example, the headache, which is a very common and early symptom, may become so severe as to be almost unendurable and this is true in cases which present no symptoms suggestive of a complicating meningitis.

Sleeplessness due to the persistent coughing is seen in some cases just as in others the patient may be unusually sleepy.

The very suddenness of the onset of the temperature in pneumonia favors the development of severe nervous symptoms in those who for one reason or another are possessed of less stable nervous equilibrium than the ordinary individual. Thus in children a pneumonia may begin with convulsions and the statement is constantly made that this is a common thing, but Schlessinger, who has had unusual opportunities to study pneumonia in children says that convulsions are rare except in the very young. The appearance of convulsions later in the course is exceptional and points to the development of some complication. It is not clear why convulsions should be exceptional in the onset of pneumonia in childhood for they are certainly common at that period of life from processes much less severe than the disease under consideration. Other diseases of sudden onset like scarlet fever, frequently cause convulsions. If the sudden rise in temperature were the only factor, the convulsions

should be common. No doubt the special products of the infecting organism is more influential in determining the character of the symptoms than is the degree of temperature.

Severe cerebral irritation at the onset is less common in adults than in other infections. Mania, for example, is rare, although in certain other diseases, notably typhoid, an acute mania is not infrequent.

Coma may initiate the disease but personally I have seen this more frequently in adults than in children, an experience however which is not ordinary.

In children one sees not infrequently the picture to which for want of a better word the name meningismus has been applied. It consists of a marked rigidity of the neck with retraction of the head, often accompanied by vomiting, delirium and some times by convulsions. This condition is frequently looked upon as a complicating meningitis and yet the symptoms listed above and the outcome do not warrant the diagnosis. The condition seems rather one of meningeal irritation than actual inflammation. Formerly there was no way of settling this question and if the case recovered it was said not to be a meningitis, while if it died, that diagnosis was regarded as sustained. Fortunately we can now safely and certainly reach a diagnosis by means of the lumbar puncture. This has shown that more of these cases are really meningitis than had been thought and that it is perfectly possible for a patient with a pneumococcus meningitis to recover.

The most common cerebral disturbance in the course of pneumonia is the delirium. This may appear at any period in the course of the disease, even in some cases developing after defervescence. There are a variety of factors favoring the development of this symptom and among them the most important are the age, the location and extent of the pneumonia and alcoholism.

The influence of age has already been mentioned in connection with the appearance of convulsions. It is apparent also in the causation of delirium which is commoner in children than in adults and in adults than in the senile. The cerebrum becomes progressively less irritable and labile as age increases and while this is not much in the way of an explanation there is no question of the fact. No doubt the more gradual onset of the symptoms and the often entire freedom from elevation of temperature in old people explains in part the slightness of the cerebral disturbances. Exceptions to the rule however are not rare and serious delirium is seen in the aged as well as in the child and the adult.

Most authors agree that delirium is more frequent and severe in upper lobed pneumonias than in those affecting the lower parts of the lungs. Thus Hensel in 117 cases of pneumonia in which the upper lobes were involved alone or in part, found 47 with severe cerebral symptoms, *i. e.*, 40.2%, while 201 confined to the lower lobes only 51 or 25.5 per cent showed similar disturbances.

The explanation of this fact is not clear, but that offered by Aufrecht appears reasonable. He

suggests that a pneumonia of an upper lobe causes a considerable reduction of the inspiratory negative pressure in the chest so that the return flow of the blood from the brain is more impeded. This obstruction to the return flow of the venous blood favors the transudation of fluid from the arterial and capillary vessels and in this way favors the development of cerebral symptoms.

It seems to me that there are some other factors which should be mentioned here. It is easy to see that an infiltrated upper lobe may exert pressure upon the superior vena cava and jugular veins and thus impede the return circulation of the blood. This slowing of the circulation not only favors the transudation of the fluids of the blood but it exposes the brain tissue to a more prolonged action of the bacteria and their toxines and at the same time lessens their nutrition by exposing them to blood overloaded with carbon dioxide and other waste products. All of these factors working together favor the development of cerebral symptoms.

Little attention has been paid to the extent of lung involvement in the causation of delirium but that it is an important factor is shown by the figures given by Sears and Larrabee. They found that 26.9 per cent of the cases involving one lobe showed delirium, 37.3 per cent of the two lobed cases, 35.4 per cent of the three lobed, 50 per cent of the four lobed and 100 per cent of the five lobed.

One lobe 590 cases, delirious 159, *i. e.*, 26.9%
Two lobes 233 cases, delirious 88, *i. e.*, 37.3%
Three lobes 99 cases, delirious 35, *i. e.*, 35.4%

Four lobes 8 cases, delirious 4, *i. e.*, 50.0%
Five lobes 4 cases, delirious 4, *i. e.*, 100%

They however give no data as to the relative amount of influence of the various lobes.

Alcoholism is a most important factor in the causation of delirium. The more excessive the use of alcohol has been the more certain delirium is to develop and yet it may and does occur in those who would not be said to be heavy drinkers, in the man who takes his two or three drinks of whisky daily and is never seen under the influence of liquor. The percentage of cases in which this factor is the determining one varies with different material. Huss in Sweden found the percentage as high as 10-12%. Fortunately the figures are not generally as high as this. Sears and Larrabee found 126 cases of delirium in 256 alcoholics, *i. e.*, 49% while only 108 cases of delirium occurred in 441 not accustomed to alcoholic stimulation. The delirium is usually that familiar as the delirium tremens but a recent case has shown me that the delirium due to the disease may simulate the alcoholic delirium perfectly. The patient was a young man, brought to the hospital without any history, suffering from a pneumonia of the right upper lobe, with delirium exactly simulating an alcoholic delirium. When however the young man had recovered he assured us that he had never used alcohol even in moderate amounts.

When venesection was employed very generally in the treatment, delirium was more frequently observed than now. It is reasonable to suppose that this artificially produced anaemia has the same pre-

disposing influence upon delirium that any pre-existing anaemia has.

Another rare but interesting cerebral symptom is the hemiplegia, monoplegia or aphasia appearing any time in the course of the disease and proving either by the prompt recovery or by the autopsy, that it is not due to any demonstrable organic change. The cases are apparently of two sorts. One occurs in old people with atheromatous changes in the blood vessels and are comparable to the intermittent claudication seen in old people. So long as their condition is the usual one and they are not subjected to either mental or physical strain the circulation in the brain is sufficient. When, however, the heart has to do the additional work put upon it by the intercurrent pneumonia, the brain suffers and hemiplegia or some other focal cerebral symptom develops. Exactly the same thing occurs in old people quite commonly as the result of mental excitement. The other group of cases, occurring in the young, especially in children, are not so easily explained, but the explanation offered a few pages back in regard to the frequency of delirium in upper-lobed pneumonias probably holds here, for the great bulk of the cases are seen in the course of pneumonia of the right upper lobe. Personally I have seen but one instance of such transient paralysis in the young. It was a child of three who presented a paralysis of the arm lasting about thirty-six hours and appearing in the course of a right upper lobed pneumonia. The recovery from the paralysis was

complete in thirty-six hours and from the pneumonia in the usual time.

Of late attention has been drawn to the condition of the pupils in the course of penumonia. Zanoni in a report covering some hundred cases notes from the onset of the disease that the pupils are larger than normal and that the pupil corresponding to the affected side is the larger. This same thing has been noted by others, but with nothing like the frequency found by Zanoni. The phenomenon is explained upon the basis of a reflex irritation of the upper cervical sympathetic ganglion. This dilation of the pupil may appear early, even before physical signs do or may be delayed even until some days after the crisis. Zanoni believes that a failure or early disappearance of the mydriasis is an unfavorable prognostic sign.

Another pupillary phenomenon noted by Schultze is sluggish reaction or even absence of the reaction of the pupil to light without there being any reason to believe that there is a meningitis.

In connection with these pupillary changes we may speak of the flushing of the cheek of the affected side. This was formerly believed to be a constant and diagnostic sign, but broader observation shows that such flushing is often absent and if present is often on the opposite cheek. Eason believes that the flushing is due to irritation of the vasodilator nerves which pass through the first to the fifth dorsal nerves to the lower cervical ganglion. He also holds that the pallor of the cheek, with or without widening of the pupil of the affected side is due to an irri-

tation of the vaso-constrictor fibres in the second to the fourth dorsal nerves.

The pupillary changes and the flushing or paling of the cheeks are seen much more often in apical and upper lobe pneumonia than in other cases.

Pfaundler found an absence or reduction of the knee jerk in 55 of 200 children, under ten years of age, suffering with pneumonia. These cases all showed evidences of a severe intoxication, and particularly the cerebral symptoms of such intoxication. This loss of the knee jerk is not at all a diagnostic sign of pneumonia and throws light rather upon the severity of the process than upon its nature. The pupillary changes, however, may sometimes be of some value as pointing to the location of the process. When we recall that the diagnosis of upper-lobed pneumonias is generally more difficult than when the lower parts of the lungs are involved, every little thing may be of importance.

Urine. Most of the changes shown by the urine in pneumonia are seen also in other infective processes. The amount is decreased to one-half the normal, although in some cases a decrease to 400 or 500 cubic centimetres occurs. Complete anuria is very rare. The diminution in the amount of the urine continues throughout the disease, to be followed after the crisis by a transient, epicritical polyuria. The specific gravity of the urine is high, 1,025 to 1,030, although higher figures than the latter are occasionally seen. The urine is darker than normal and highly acid. This acid reaction continues throughout the course, but Pick has noted in a considerable proportion of cases that the urine becomes

neutral or even alkaline about 24 to 48 hours after the crisis and remains so for a day or more.

Upon standing and cooling the urine deposits an abundance of reddish deposit consisting mainly of the acid urate of sodium and a few crystals of uric acid.

The amount of urea is almost invariably increased and this too though the patient is usually upon a low diet. The excretion for 24 hours may, according to Brattler and Vogel, reach 70 grams. In many cases there is a still further increase during the period of resolution. This is partly the result of the increased amount of the urine, but is mainly due to the absorption of the large amount of the nitrogenous material contained in the exudate into the lungs.

The amount of uric acid also is increased.

Much interest has been given to the excretion of the chlorides in the urine and the subject has been especially studied by Hutchinson. The chlorides in the urine are decreased to a marked degree in almost all febrile disease, but in none is the decrease so great or so constant as in pneumonia. In this disease they may entirely disappear although in general the diminution is not so great as this. Such complete absence may continue for a day or two or even last throughout the course of the disease, and if transient their disappearance does not necessarily coincide with the height of the disease. The average excretion is two grams per day instead of the normal 15 grams (Neubauer and Vogel). There is no relation between the degree of temperature and the fall in chloride excretion. We see high temperature with low excretion or high excretion and the same is true of the low temperatures. The amount of pul-

monary involvement also has no apparent bearing. Thus in two of Hutchinson's cases the lung area involved was very small and yet there were no chlorides in the urine. In other cases with large exudation the chlorides were present and in others in which the amount of pulmonary exudate progres-

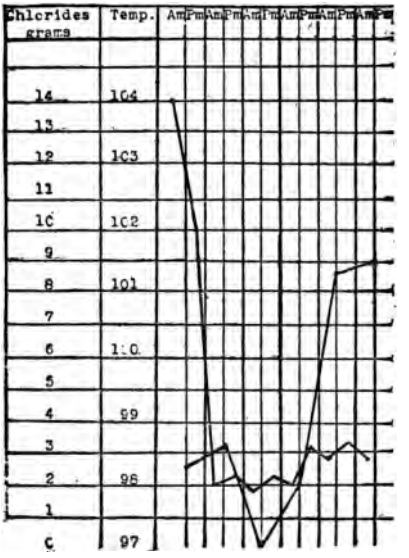


CHART X. Relation of chlorides excreted in urine to temperature at and subsequent to the crisis. (From Hutchinson.)

sively increased, there was no parallel change in the excretion of chlorides. Moreover the decrease in the chlorides appears early in the disease as is illustrated by a case of von Noorden's in which the excretion fell to 2.2 grams in the first 24 hours.

The chlorides may reappear in the urine with

great suddenness, being absent one day and present in normal or nearly normal amounts the next. Usually such reappearance is delayed for about two days after the crisis.

What becomes of the chlorides during their disappearance has been and still is a matter of discus-

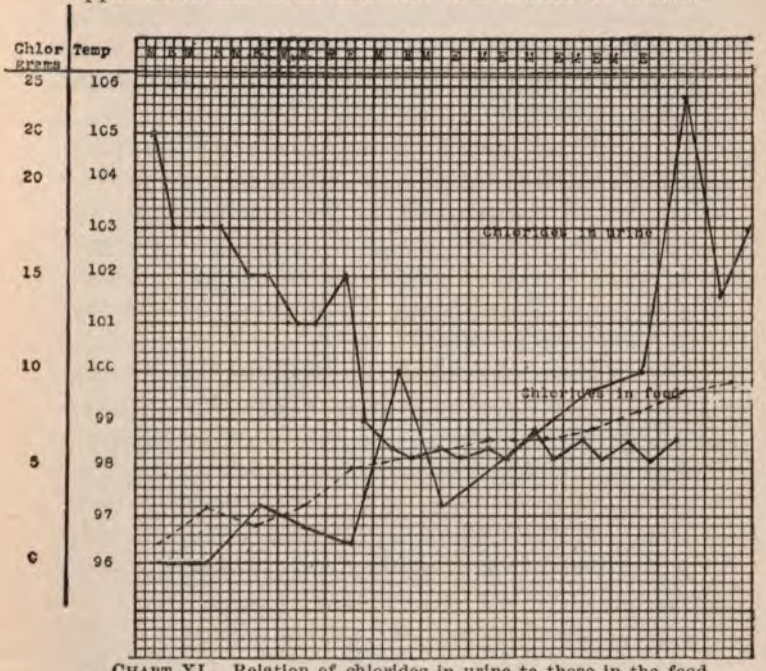


CHART XI. Relation of chlorides in urine to those in the food and to temperature. (From Hutchinson.)

sion. It has been suggested that they are excreted in the sputum and while pneumonic sputum really does contain more chlorides than in other conditions the excess is not marked and the total amount of sputum is small. That the chlorides do not accumulate in

the pneumonic area is shown by the fact that analysis of such areas show an average of 1.69 per cent of sodium chloride while other lungs show 1.4 per cent; too slight a difference to account for much. Another suggestion is that the chlorides are not absorbed from the intestinal tract, an idea which is done away with by Röhmann who by weighing the chlorides in the food and that excreted in urine and faeces found that the absorption of the chlorides was perfect. Von Limbeck and Moraczewski have shown that the blood of pneumonic patients contains less than the normal amount of chlorides.

Effort has been made to give the excretion of chlorides a prognostic significance, the outlook being the better, the greater the amount of chlorides in the urine. Unfortunately there seems to be no grounds for this idea.

The decrease in chlorides may be of considerable value in making a diagnosis. While one can infer nothing from the mere coincidence of temperature and a small amount of chlorides in the urine because this combination is met in almost all fevers, a temperature with a normal or considerable excretion of the chlorides would speak strongly against pneumonia.

Croupous pneumonia is the only disease of the lungs accompanied by a low excretion of the chlorides and such low excretion with a pulmonary process speaks for a pneumonia. For example if a case of tuberculosis develops an acute pneumonia with low amount of chlorides, the inference would be that the pneumonia was not due to the tuberculosis, but to an added pneumococcus infection.

There are as yet no data upon chloride excretion in pneumococcus infections which do not involve the lungs, but from such facts as we know it seems likely that the decrease of the chlorides is the result of the action of the pneumococci rather than of their location.

The excretion of phosphates and sulphates varies, according to some being increased while others have found them decreased.

Albumin appears in the urine at some time in the course of most cases, indeed if all the urine is examined some albumin will probably be found in all. The figures given vary over rather wide ranges. Rosenstein found it in 23 per cent of 130 cases. Cramer in 45 per cent of 66 cases. McCrae found it either with or without casts in 47.7 per cent of 450 cases. Sears and Larrabee report still larger percentages ranging from 75 to 100 per cent, and in a general way more frequent in bi-lobar and tri-lobar pneumonias than in the mono-lobar cases; Norris in 71 per cent of 455 cases usually without but often with casts.

Casts, hyaline and granular, as already indicated, are by no means rare. Norris found them in 249 of 455 cases, and in four of these there was no albumin in the urine. Since the introduction of centrifugal precipitation of the urine the frequency with which casts are found has greatly increased and the 50 per cent of cases reported by Norris corresponds to about average experience.

In spite of the great frequency with which albumen and casts are found in the urine, nephritis, as will be pointed out when speaking of the complica-

tions, is rare. Changes in the kidneys of fatal cases are practically always found: Fränkel and Reiche, in 61 autopsies upon pneumonias, report them as almost always confined to the cortical layer. The secreting epithelium was mainly affected and in all cases, though to a varying degree, there was an exudate in the glomerular capsules.

In the cases in which a jaundice is added to the pneumonia the bile pigments can be found in the urine.

The **gastro-intestinal** symptoms are in no way peculiar to this disease, being only such as are seen in all febrile disturbances. The appetite fails and when the dyspnoea is considerable the taking of food is still further disturbed, the air hunger being so marked that it overcomes any disposition to take food. In a small proportion of the adult cases nausea and vomiting appear as initial symptoms, but only rarely do they continue for any length of time. If the cough is persistent it may excite vomiting here as it does in other diseases. In a few cases the vomiting is cerebral in origin and due either to cerebral irritation or to the development of a complicating meningitis. In children vomiting is a much more frequent initial symptom than in adults.

The digestion is enfeebled but unless the diet is inappropriate or the stomach is injured by injudicious and excessive drugging, the patients rarely suffer from indigestion during the course of the disease.

Constipation is the rule, but in some cases diarrhoea appears and may become a serious complication.

The liver shows changes in only a small proportion of the cases and then only in the way of a jaundice to which reference will be made later.

The statements in regard to the spleen vary. Juergensen, for example, says that he finds the spleen enlarged more often than not. So far as my personal experience goes an enlargement sufficient to enable one to palpate the spleen is exceptional and the figures given by McCrae, 30 times in 450 cases, appear to me to be about correct.

CHAPTER VI.

COMPLICATIONS AND SEQUELÆ.

Pleurisy. This is by all odds the commonest complication, so common indeed, that it might perhaps be better to regard it as a normal part of the disease than as a complication. In all cases in which the pneumonia extends to the surface of the lungs, and this is practically all of them, an inflammation of the pleura results. Such pleuritis has no effect upon the clinical course of the pneumonia and usually causes such insignificant symptoms and physical signs that they are entirely overlooked in the attention given to the pneumonia.

The site of the pleurisy varies with the site of the pneumonia, and while the fluid usually accumulates in the lower and posterior part of the general pleural cavity it may be encysted in the interlobar fissures, the course of which has been previously described, or between the lung and the mediastinum or diaphragm. The physical signs of such encysted fluids do not differ from those of exudate in the general cavity, but their location sometimes leads to confusion and error in diagnosis. Usually the pleurisy ends with the primary disease and leaves no effects. Some pain is always present and even in cases which take a favorable course may be the most

distressing symptom. The character of the patient has no doubt some bearing upon the suffering caused by the pleurisy, but my personal experience leads me to fear the cases in which the pain is much beyond the ordinary, because of the frequency with which I have seen empyema result in these cases.

The only physical sign caused by the ordinary degree of pleurisy is the friction. This is often of very short duration so that in many cases it is never heard or heard only at a single examination.

Just what degree of pleurisy warrants one in calling it an actual complication is largely a matter of opinion and this is probably the reason for the wide range in the figures given by various authors as to the frequency of pleurisy. The reports from the Vienna Hospitals quoted by Juergensen give 298 cases in 5,738 cases of pneumonia, *i. e.*, 5.2 per cent; Huss of Stockholm found 104 in 2,616, *i. e.*, 4 per cent; Fismer of Basel found 35 in 230,—15.3 per cent. Aufrecht in 1,501 cases had 59 serious pleurisies and 24 empyemas, making 5.5 per cent; Sello reports 65 in 750 cases, *i. e.*, 8.7 per cent. Grisolle places the frequency of pleurisy at 12.6 per cent, while Dinstl placed it at 15.8 per cent. Sears and Larrabee record 53 cases of pleurisy with demonstrable fluid in 949 cases, *i. e.*, 5.5 per cent. Norris reports 12 pleurisies with effusion and 6 empyemas in 500 cases—3.6 per cent. Townsend and Coolidge have 17 in 1,000 cases—1.7 per cent. Meyer has 20 pleurisies with effusion and 12 empyemas in 500 cases—6.4 per cent. Adding these together we have 13,784 cases of pneumonia with 705 pleurisies, *i. e.*, 5.1 per cent. The figures of the various reports

range from 1.3 per cent to 15.8 per cent. Maragliano employed the exploratory puncture in 58 cases of pneumonia and found a sero-fibrinous or fibrino-purulent exudate in 38 cases—65.5 per cent. In contrast with these clinical data is the report of Kerr of 171 autopsies upon lobar pneumonia with 118 cases of pleurisy—69 per cent, the character of the exudate being as follows:

Acute fibrinous pleuritis	74
Acute serofibrinous pleuritis	38
Acute purulent pleuritis	6

It seems reasonable to say that if the pleurisy causes physical signs, other than mere friction, which permits of its recognition, it should be regarded as a complication, and when we recall that at least 400 cubic centimeters of exudate in the pleural cavity must be present before physical signs marked enough to permit of certain recognition, this seems the more reasonable. The presence of such an amount of exudate must have some effect.

The pleurisy may precede or follow the pneumonia, the former being the decided exception. In 57 cases in which Sello demonstrated the presence of fluid by exploratory puncture, only two were at the beginning, 31 during the pneumonia and 24 after the crisis. It usually appears about the fourth or fifth day of the disease.

The physical signs due to an exudate in the pleural cavity do not require description here, but some words of caution may be in order. One must not be too ready to diagnose a pleurisy with effusion in addition to a pneumonia upon a single examination. The intense dulness, the loss of vocal fremitus, of breath

and voice sounds, physical signs which are of major importance in the diagnosis of a pleural exudate, may all be found in cases of pneumonia. All that is required is to have the bronchi of the pneumonic area plugged to produce all these signs. If then one finds such signs in a patient whose illness suggests by its mode of onset and by its other symptoms a pneumonia, one should wait and re-examine the patient before making a diagnosis. It may require a day or two before a definite diagnosis is reached. In cases such as these especial attention should be paid to the presence or absence of evidences of pressure and its effects upon the thoracic and abdominal viscera. If the area of dulness under consideration is small, reaching, let us say, to the angle of the scapula, displacement of the viscera need not be looked for. But when the dulness indicates a yet larger effusion we may expect pressure effects, and the more massive the exudate the more certain we are to find them. The effects of effusions into the left thoracic cavity are much greater than those caused by right pleural effusions. An exudate of 500 to 600 c. c. in the left cavity will displace the apex of the heart to or nearly to the left border of the sternum; one of 1,200 c. c. will displace it to the right border of the sternum and one of 1,800 or 2,000 will put it nearly or quite to the right nipple line. An exudate of 2,500 c. c. in the right cavity will not displace the heart to the left more than one or at most two centimeters. If then we find a case in which the question arises as to a differential diagnosis between a massive pneumonia and a pleurisy with effusion, the absence of displacement of the

heart, liver or spleen speaks for a pneumonia, while distinct evidence of displacement of these viscera shows the case to be one of pleurisy with effusion.

In some cases, however, in spite of every effort and of repeated examination the diagnosis will still remain obscure and resort must be taken to the exploring syringe and needle. This instrument is extremely useful in determining the presence of fluid, but its greatest value is in enabling us to learn the nature of the fluid, an important fact which would otherwise escape us. I cannot refrain from saying that most practitioners use the exploring syringe far too infrequently. Its use requires no special training and if one makes the puncture with due attention to cleanliness the puncture is safe and painless.

A question which arises far more frequently than the differentiation of a pneumonia from a pleurisy is whether a considerable exudate is present in addition to a pneumonia. Here as in the case just mentioned one must pay more attention to the pressure symptoms such as the cardiac displacement or the downward displacement of the diaphragm and consequent reduction in the size of Traube's space than to the dulness or the changes in breath or voice sounds. In such cases the possible combinations of physical signs are very numerous and are especially puzzling to one who expects to find at the bedside the reproduction of the average physical signs as described in the text books. Here also it may be necessary to resort to the exploratory puncture. Not infrequently one will fail to get any fluid on puncture, although morally certain that there is an exudate in the cavity. The reason for such failure is clear when

one recalls that the pleural exudate in such cases is often almost pure fibrin and that when there are considerable masses of fibrin in the pleural cavity the resulting physical signs are just such as occur with fluid.

The pleurisy even when it is well enough developed to permit of its certain recognition has as a rule but little effect upon the course of the pneumonia. The cases end by crisis or lysis and the exudate undergoes resorption and things are as they were before. The mortality is not materially increased by the pleuritis. This accords with most authors, but Fismer of Basel in a rather short series of cases found the mortality in cases complicated by pleurisy double that of the average mortality, 40 per cent instead of 16.5 per cent. In 88 cases there were 21 deaths, giving a mortality of 23 per cent.

In most cases there are no after effects, but in a few the adhesions formed between the visceral and parietal pleura cause trouble. Usually this is nothing more than pain, which is influenced by exertion, by deep breathing and according to the patients' statements, by changes in the weather. Gradually in the course of months this all disappears. In other cases the adhesions are so thick or become so thick that their inevitable contraction deforms the chest.

Empyema. Lastly we must consider the development of an empyema out of a pleurisy. It is interesting to note that the pneumococcus which very rarely causes suppuration in the lungs does so rather frequently when it attacks serous surfaces. The fre-

quency with which empyema occurs varies from time to time and place to place.

Aufrecht	1,501	cases—	23	empyema	= 1.5%
Sello	750	"	—34	"	= 4.5%
Sears and Larrabee	949	"	—19	"	= 2.0%
Norris	500	"	— 6	"	= 1.2%
Meyer	500	"	—12	"	= 2.4%
Total	4,200	"	—94	"	= 2.2%

In a general way an empyema is more apt to follow a severe than a mild pneumonia and as previously stated is especially to be feared if the early pleural inflammation causes intense pain. It seems reasonable to suppose that in these cases the pain is in proportion to the severity of the pleurisy and that suppuration is more prone to follow a severe than a mild pleuritis. Cases which end by crisis are not often followed by empyema, but they may be. The infrequency of this is shown by Sello who found only one instance of a critical ending of the pneumonia in 34 cases of empyema. Age has some influence, for Netter in 286 collected cases of empyema found 215 under 30 years of age. For some unknown reason empyemas are considerably more common in the left than in the right pleural cavity, a fact which is still more striking when we recall the greater frequency of right-sided pneumonia.

The development of an empyema does not cause any characteristic symptoms. The temperature of the pneumonia has fallen by lysis and for a few days sometimes even for a week or two the patient seems to be progressing favorably. Then the temperature

begins to go up again gradually and follows usually an altogether irregular course although it may be continuous, intermittent, remittent or hectic. Chills either at the onset or during the course are exceptional so long as the pleural infection is due to the pneumococcus only. In some cases there is no elevation of temperature at all. Sweats, particularly at night, become frequent and are troublesome and exhausting. A leucocytosis, often a very marked one, is usually present and a persistent leucocytosis or one appearing after the crisis of a pneumonia, should, even though other symptoms are wanting, suggest the possibility of an empyema and lead to a most thorough examination of the chest. Symptoms referable to the chest are often wanting. There is no pain and dyspnœa is rare except when the amount of exudate becomes extraordinarily large. Such symptoms as these coming on after a pneumonia should automatically suggest to one the possibility, even the probability, of an empyema, but the diagnosis can be made only by the physical examination of the chest and the use of the exploring syringe. The physical signs are those of fluid in the pleural cavity and usually they appear over the lower and posterior part of the chest, but they may appear elsewhere, the fluid being encysted by adhesions either in the general pleural cavity or more often in the interlobar fissures or between the lung and the mediastinum. Wherever the fluid accumulates the signs are the same, and no matter what the general symptoms may be, the exploring syringe should be employed to obtain some of the fluid for examination. Where the needle shall be inserted is de-

termined by the physical signs. Oftenest these will lead one to insert the needle in the 6th to 8th intercostal space in the posterior axillary line or the scapular line, but occasionally when the fluid is encysted the puncture must be made higher up or further forward. If the indication for exploratory puncture exist, one should not be discouraged by failure to obtain fluid at the first trial. It may be necessary to insert the needle in two or three places, either at one or several sittings, before fluid is struck.

The fluid of a pneumococcus empyema is a thick, greenish yellow, odorless pus, the laudable pus of our forefathers. It is rich in cellular elements. The cells are almost exclusively of the polymorphonuclear type, the mononuclear cell and the endothelial cells being few in number. The bacterial content of the fluid is much more important than the cellular. The organism most often found is the pneumococcus alone. In some cases it is associated with one or more varieties of the ordinary pus cocci and sometimes the latter only are found. The admixture of pus cocci is a serious matter, for such cases take a much more active course than that shown by the pure pneumococcus cases. The temperature is higher, more irregular, chills are more common and the sweating is more severe. Edema of the chest wall, exceptional in the pure pneumococcus cases, is not rare in this sort. The bacterial content of the fluid has also a bearing upon the prognosis and treatment. The pure pneumococci cases are mild. They not infrequently end spontaneously either by rupture into the bronchi and subsequent closure of the cavity or by absorption of the pus. Aspiration of the fluid is

often the only surgical procedure necessary. This is due to the fact that the pneumococci rapidly lose their viability so that the fluid becomes sterile. It would, however, be too much to say that the pure pneumococcus empyemas necessarily run a mild course. They may become very destructive indeed and one must be prepared to treat them as actively as one must always treat the streptococcus empyemas if the aspiration proves insufficient.

When the pus cocci are present the prognosis is much less favorable and while spontaneous recovery is possible the chances of it are so small that we have no choice and must drain the cavity.

The post-pneumonic empyema more frequently than any other form, ruptures into the bronchi. According to Netter this happens in about 25 per cent of the cases. It rarely happens before the twentieth day or after the sixth week. It is followed by the expectoration, in the course of a few minutes or hours, of a considerable amount of pus, the abundance of the sputum being in marked contrast to the small amount existing previous to the rupture. From then on this free expectoration may continue but frequently there follows a period of freedom from expectorate until the cavity is refilled, when again there is a spell of profuse expectoration.

Abscess. Because of the similarity in the symptoms of abscess of the lung and those of empyema it seems natural to consider here this fortunately rather exceptional ending of a pneumonia. In 424 autopsies by Aufrecht and Kerr there were 8 abscesses in the lungs, that is, in 1.9 per cent of the cases going to autopsy. Sello in 750 cases found 11,

which is 1.5 per cent. Only the most general statements can be made as to the cause of this ending of the pneumonia infiltration. It occurs in those who are debilitated for any reason and in alcoholics. Both Juergensen and Eichorst state that suppuration is more common in the upper than the lower lobe, but personal experience would lead to an opposite statement and Suppier says that 80% of abscesses of the lung are in the lower lobes and that of 49 cases upon which he operated, 23 were subsequent to croupous pneumonia.

During the early course of the attack of pneumonia there is nothing to lead one to anticipate a pulmonary abscess except that one is always looking for trouble when pneumonia appears in the alcoholic or debilitated and especially so if the infiltrate is large. The disease runs its course with the temperature usually falling by lysis, for this complication like the empyema rarely develops in cases ending by crisis. The temperature returns to or almost to normal, but the general condition of the patient does not improve. The temperature usually goes up again and takes an irregularly intermittent or remittent course, rarely interrupted by chills but often by sweats. The leucocytosis, if it had disappeared, begins again. Thus far the picture is just that seen with an empyema and this is the thought naturally suggested by such a course of events. The physical signs however are different in that they are usually absent. When present their nature is determined by the size and site of the abscess. If the abscess is of considerable size and located near the surface of the lungs, the signs are those of

a solid mass containing no air, *i. e.,* a dulness which usually cannot be sharply outlined by percussion. The breath and voice sounds over this area are absent as is also the vocal fremitus. These it will be noted are the same physical signs presented by an empyema. It is only when the abscess does not reach the surface of the lung and yet lies near enough to it to change the percussion note on heavy percussion and while not affecting the note of light percussion that abscess of the lung may be reasonably suspected from the physical signs.

When the constitutional disturbances usually associated with pus formation, appear after an acute pneumonia and the physical signs of an empyema are wanting and there is no reason to think of a complication elsewhere than in the lungs, a pulmonary abscess may be reasonably suspected. In general however the diagnosis is not made until the changes appear in the sputum. Usually this occurs suddenly. The patient who has been raising, often with persistent and painful effort, a small amount of muco-purulent sputum suddenly expectorates a considerable amount of pus. The amount is determined by the size of the abscess and ranges from a few up to several hundred cubic centimeters. The sputum is frankly purulent, yellowish or creamy in color, though it may be colored with blood pigments or if infected with chromogenic bacteria may assume other colors as green or blue. It is not particularly offensive at first though it may become so later. Microscopically it contains pus cells, epithelial cells, crystals of various sorts, bac-

teria and almost always shreds of lung tissue, sometimes even macroscopical pieces. The appearance of these large pieces and shreds are of importance for they not only indicate an acute destruction of lung tissue but they enable one to distinguish the pulmonary abscess from an empyema rupturing into a bronchus.

The general condition of the patient always improves after the rupture of the abscess and in a certain number of cases, the improvement continues and eventually complete restoration indicates that the abscess has healed. In other cases, however, pus continues to form and a set of symptoms and physical signs develop which are highly characteristic.

The patient learns that in certain positions, he is freer from cough and expectoration than in others. Usually this is lying upon the affected side or sitting upright. In this position he is comfortable for long periods, a few hours or in some cases even a few days when he begins to cough again and in a few minutes brings up a large amount of pus. Then follows another period of freedom which may however be interrupted at any time by assuming the right position. The explanation of the periodicity of cough and expectoration is that so long as the patient is in a position which places the bronchus entering the abscess high up in the wall, the secretion accumulates and does not irritate the bronchial mucous membrane. When the cavity is full or when the patient's position is so changed that the bronchus enters below the level of the accumulated secretion, cough and expectoration comes on. If the

drainage of the cavity is free and sufficient the general health of the patient may be quite restored although eventually the organs may undergo amyloid changes.

If the abscess cavity is large enough and superficial enough its alternate filling and emptying gives an interesting play of physical signs. Dulness with absence of breath and voice sounds when the cavity is full, replaced immediately after the emptying with tympany, with Wintrich's change, amphoric breathing and râles of varying size and timbre.

The abscess does not always empty into a bronchus but may rupture into the pleural cavity, causing an empyema or a pyo-pneumo-thorax. It may rupture into the oesophagus, the pericardium, into the abdominal cavity or some of its viscera and cause a series of symptoms which do not require mention here.

Gangrene. Still less frequently than the abscess, we see gangrene follow the pneumonia, far less often than we see pneumonia follow gangrene. The causes which underlie the gangrene of a pneumonic lung are as obscure as those of the abscess. Patients exhausted by previous disease, by generally unhygienic living, by chronic alcoholism, by diabetes, suffer more frequently than those otherwise healthy. Age has some influence apparently, for the average age in Hensel's fourteen cases was 47 and the extremes 28 and 82. One who suffered from a putrid bronchitis before the onset of the pneumonia is apt to have a gangrene follow. The infrequency of this complication is clearly shown by Aufrecht's

statement that he had no case in his series of 1501. Hensel in 5072 autopsies found gangrene of the lungs 83 times and 14 of these followed fibrinous pneumonia. Kerr in 171 autopsies on pneumonia found gangrene twice.

Much that has been said of the symptoms and course of the pulmonary abscess is true of the gangrene. It cannot be anticipated, coming on often when least expected. The temperature takes the same irregular course seen in the abscess and the nature of the complication is first suspected when the sputum takes on its characteristic peculiarities. The sputum becomes stinking and the odor is so penetrating and all-pervading that the patient must often be isolated. The sputum loses its odor very quickly on standing though the odor reappears on shaking. The amount of the sputum is often excessive, even as much as 1000 c. c. per day. It is of a dirty gray to yellow color, and on standing settles into three layers, an upper frothy layer, a middle layer consisting of dirty gray or greenish serum and a bottom layer of granular looking material. In this layer are found the bits of lung parenchyma which are so important as evidence of actual destruction of tissue. The size of these particles varies from microscopical to several centimeters in diameter. They may be pale gray to black in color and microscopically show traces of the alveolar structure of the lungs. In addition to these we find pus cells, red blood cells, haematoidin crystals, fatty acid crystals, bacteria of various kinds, yeast, and sometimes infusoria.

With the expectoration of the lung tissue the

signs of the pneumonia are gradually replaced by the signs of a cavity. The cough which at first is most distressing is gradually replaced by one which is intermittent and influenced by position, exactly as sketched when speaking of the pulmonary abscess.

Empyema or pneumothorax is more to be feared than the abscess and there is especial danger of haemoptysis, which is a common event in these cases. The amount of the haemoptysis varies with the size of the eroded vessel and ranges from a mere blood trace in the sputum to a fatal hemorrhage. The bleeding may occur once or many times.

The duration of life after the onset of a post-pneumonic gangrene is usually not long. The patient may die from gradual exhaustion or from some rapidly fatal complication. Death however is not inevitable for in a small percentage of the cases recovery takes place, the walls of the cavity becoming adherent to each other thus obliterating the cavity. In still other cases the cavity remains and patient continues for years to expectorate a stinking purulent sputum.

Delayed Resolution. In contrast to the destructive changes in the pneumonic area we find delayed resolution and organization. The dividing line between these two is altogether indefinite and some, Aufrecht, for example, are unwilling to admit that there is any distinction between them. Leyden on the other hand is quite certain that the division is justified by clinical experience and Fraenkel agrees with him.

Just what constitutes delayed resolution must be

somewhat a matter of opinion for the time required in the average case of pneumonia varies. The exudate may entirely disappear from the lungs in as short a period as twenty-four hours but this is so exceptional that it cannot be considered in forming an opinion of the average time required for resolution. Usually several days are required but every one has seen cases in which even after the passage of days and weeks the signs of consolidation still persisted over the lungs. Even after the passage of these weeks perfect resolution is possible but at any time the evidences of organization may appear.

The causes for such delayed resolution and organization are still obscure. While some of the patients are old and debilitated by disease or alcoholism, others are young and previous to the onset of the pneumonia have been well and strong. Marchand and Kahlden believe that a previous attack of pneumonia which did not entirely clear up but left behind it thick pleural adhesions and probably some thickening of the lung tissue itself just beneath the pleura, retards resolution and favors organization. It is supposed that in the pleural adhesions there is an abundant collateral circulation of blood vessels which increases the amount of blood going to the pneumonic area and thus favors the organization of the exudate. There can be no question of the fact that a very considerable proportion of the cases of organization do occur in patients who show at the autopsy old and firm pleural adhesions, but not all cases do. Thus one of Fraenkel's cases showed at the autopsy that there were no such

changes in the pleura and I recall a personal observation of such post-pneumonic organization appearing in a young boy who had never had any previous illness, and whose chest gave no evidences of the presence of pleural adhesions. It would, however, seem reasonable to believe that such adhesions do favor the organization of the exudate but less because of the blood supply in them than because they lessen the motility of the chest wall.

Eppinger speaks of the influence of this factor. How much the movement of the chest wall influences the absorption of the exudate in the lungs cannot be measured but it certainly is an important element. Recently Marchand has drawn attention to another element in these cases. On the basis of personal observation and eleven reported cases of indurative pneumonia accompanied by autopsy reports, he expresses the opinion that pericarditis, which was present in all these cases, is an important factor in the non-absorption of the exudate. The character of the pericardial exudate is unimportant, for eight times it was purulent, four times cloudy and twice sero-fibrinous.

Fraenkel because of the fever present in most cases undergoing organization believes that there is an infective agent active in these cases. Whether it is a mixed infection or not cannot be stated for lack of sufficient data.

The clinical course of this group of cases is as follows: During the active period of the pneumonia the cases present only the average picture although Leyden noted in some of his that the dulness was unusually intense and that fewer râles were present

than in the average case, evidences that the infiltration was greater than usual.

The temperature ends by crisis or by lysis and the general condition of the patient improves, but examination of the lungs shows no change there. The dulness persists without diminution. The bronchial breathing continues as do to some extent the crepitant râles. The sputum is scanty, yellowish to green in color and most important of all contains no fibrous tissue. This group of cases shows no elevation of temperature. The condition of the lung remains unchanged for weeks or months although sometimes areas appear scattered through the consolidated lung, over which signs of resolution are found. The percussion note clears up, and the breath sounds take on an approximately normal character. Then gradually the resolution takes place and the lung tissue returns to its normal condition.

Such cases are a source of great anxiety first because the suspicion of a possible tuberculosis is always excited. In these cases of merely delayed resolution the freedom from temperature, the long continued signs of consolidation without evidences of softening as shown by the appearance of large râles, the persistent failure to demonstrate the presence of the tubercle bacilli or of fibrous tissue in the sputum, are all sources of hope and encouragement to the belief that ultimate recovery is possible.

Another source of anxiety is the possibility that the delayed resolution is the fore-runner of organization of the exudate. In cases in which this misfortune appears, the temperature after the crisis or

lysis begins again to go up and pursues an altogether irregular course. In some cases the temperature of the pneumonia passes without any interruption over into that of the organization. Fraenkel holds that temperature is always present in cases undergoing organization but that this is too absolute a statement is shown by an observation of Aufrecht and also by a case reported by me at the 1899 meeting of the American Medical Association.

The physical signs over a pneumonic area undergoing organization are for a time exactly similar to those shown in merely delayed resolution but it is not long before evidences of retraction of the new formed fibrous tissue appear. In a number of personal observations these signs always appeared within the first four weeks. This clinical fact corresponds with the pathological data supplied by Kahlden. In five such cases which he examined post mortem, and found unquestionable evidences of organization of the exudate the death occurred twice fourteen days after the onset and once each, three, four and five weeks after the initial symptoms.

The dulness persists, the breath sounds continue to have a bronchial element although not the loud, pure bronchial breathing present during the active pneumonia. Râles continue but they are fine, never large unless there is added to the induration foci which undergo suppuration or gangrene. The vocal fremitus is usually reduced because of the pleural thickening. The important changes however are in the size and motility of the affected area. The chest is sunken over this area, above if the upper lobe is

affected, below if the lower, and entire side, if the whole lung is involved. The intercostal spaces are narrowed, the chest is bent toward the affected side. The motion of this side is less than the other. The distinctness of these signs varies in different cases being proportionate to the amount of lung tissue involved and the degree of the scar-tissue contraction, and so long as the contraction continues, the deformity of the chest increases. In some cases therefore we will find only slight changes while in others they are very marked.

The heart undergoes displacement and because of obstruction to the pulmonary bed the right ventricle hypertrophies.

Finally the process comes to a standstill. The temperature returns to normal although it may do this long before the organization of the exudate is complete. The ultimate course of events is largely determined by the amount of lung tissue involved and the amount of contraction. That all cases do not end favorably but that death may occur before contraction has time to appear is shown by the autopsy reports of Kahlden.

Pericarditis. The frequency with which this complication figures in statistics varies greatly according as one consults clinical or postmortem reports. This is shown by the following tables:

Vienna	5738	cases,	pericarditis	27= 0.5%
Stockholm	2616	"	"	22= 0.9%
Basel	230	"	"	9= 3.9%
Wurtzburg.....				=0.54%
Sello	750	"	"	7= 0.9%
Sears & Larrabee	949	"	"	19= 2.%

Norris.....	500 cases,	pericarditis	=4 0.8%
Townsend & Coolidge 1000	"	"	3= 0.3%
Meyer	500 "	"	5= 1.0%
Total	12383		96=0.77%

These figures show a wide range from 0.3 per cent to 3.9 per cent. Such a range in the clinical diagnosis can be easily explained by a variety of factors but the post mortem reports also show a considerable range as follows:

Osler, 100 autopsies, pericarditis.....5= 5%
Banti, 100 autopsies, pericarditis..... 5.4%
Netter, 100 autopsies, pericarditis 8.0%
Kerr, 171 autopsies, pericarditis.....26=15.8%
Stevens, 120 autopsies, pericarditis ....13=10.8%

The wide difference between the 5 per cent of Osler and the 10 and 15 per cent of Stevens and Kerr suggests that there must be factors which influence this complication as mysteriously as others are influenced. That the frequency varies in different places and at different times must be accepted as a fact, the explanation of which must be deferred to some later period.

The pericarditis is usually due to the direct extension of the inflammatory process from the neighboring lung or pleura to the pericardium, though in some cases the bacteria are transported through the blood rather than through the lymph channels. The importance of extension is shown by a consideration of the site of the pneumonia in the cases thus complicated. Juergensen says that in the great majority of his personal observations there was

a pneumonia of the lingual process of the left upper lobe. Kerr's report shows a different distribution of the pneumonia. In all but two of his twenty-six cases one lower lobe was involved and in both of these there was a pneumonia of the right middle lobe. The left lower lobe was involved in twelve cases, the right in nine and both in three. In all but four of the cases there was also pleuritis and in four there was also an endocarditis.

Dietl is of the opinion that venesection has a marked influence upon the frequency of pericarditis and meningitis basing his belief upon the following figures: In 17 fatal cases treated by venesection he found meningitis three times and pericarditis five times, while in 22 fatal cases treated by tartar emetic these occurred but once and in 14 fatal cases treated by dietetic measures they did not occur at all. In other words he found pericarditis in 29 per cent of the fatal cases treated by venesection.

Pericarditis may either precede or follow the pneumonia, but the great majority of cases develop during the height of the disease, a fact which sufficiently explains the frequency with which this complication is overlooked at the bedside. Here as elsewhere the recognition of the pericarditis depends entirely upon physical signs. It causes no subjective disturbances which would in any way suggest its onset. Nor does it disturb the clinical course of the pneumonia in any characteristic way.

In this disease, as in all the others in which pericarditis frequently appears, one should be constantly on the lookout for it and yet no matter how great the care, the clinician will be frequently cha-

grined to find on the autopsy table a pericarditis which was not even suspected. The presence of changes in those parts of the lung which border upon the heart, the friction of the neighboring pleuritis, the rapid and often noisy breathing of the patient make a satisfactory examination in many cases an impossibility. Of the various physical signs of pericarditis the friction is the most reliable in these cases, and the one upon which the diagnosis is usually based. In some cases the change of the shape and size of the cardiac dulness will give the clue to the trouble. In this connection an interesting error might be recorded. The patient was a Greek, brought to the hospital without history and unable to give any himself. Examination showed an area of dulness beginning to the right of the right nipple line, curving up across the sternum at the level of the upper border of the third rib and then down to the anterior axillary line on the left. The apex beat was easily seen about one and a half inches to the right of the left border of the cardiac dulness. Over the dull area there were no breath sounds or voice sounds or friction. It was thought to be a pericarditis with effusion, although at the time it was pointed out that a pericardial dulness of such width ought to reach higher on the sternum. The patient died three hours after entrance and the autopsy showed not a pericarditis but a pneumonia of each lung in the portions bordering on the heart and with the bronchi plugged. Such a combination of circumstances is not apt to occur again, but the case is instructive in showing a possible source of error.

If the patient survives the acute stage we may find conditions much as they may be with a pleurisy. If the exudate is absorbed, well and good, but if the signs of fluid in the pericardium persist the exploring syringe should be employed in order to learn the character of the fluid. Here as with the pleura, there is no inevitable relation between the character of the fluid and the constitutional disturbances. There may be a high and irregular temperature with a serous exudate but more frequently we find a normal temperature with a purulent accumulation.

The onset of a pericarditis in the course of a pneumonia is always a serious matter for it has considerable influence upon the mortality. While it is too much to say that it indicates an unusually severe infection, it is certain that the pericarditis mechanically influences the heart's action and increases the danger both of insufficiency from this cause and of myocarditis. Sears and Larrabee in 19 cases of pericarditis found 11 ending in death, a mortality of 58 per cent. Norris lost three out of four. Townsend and Coolidge, two out of three, and those cases which recover from both the pneumonia and the pericarditis, must be carefully watched for evidences of an obliteration of the pericardial sacs with all its possible consequences.

Endocarditis. That endocarditis may appear as a complication of pneumonia has been known for years but its frequency has, I believe, been underestimated. This is certainly true clinically for like the pericarditis, the diagnosis during life is difficult because of the severity of the primary disease and more particularly because in a great many of the

cases life does not continue long enough to permit of the development of physical signs which render the diagnosis possible.

The frequency with which this complication appears cannot be positively stated and it is likely that with this as the other complications the percentage varies from year to year and place to place. Aufrecht says that he found but a single instance in 1501 cases, *i. e.*, 0.06 per cent. Fismer found it twice in 230 cases, *i. e.*, 0.9 per cent. Wells in 6050 collected cases found 19 of endocarditis, *i. e.*, 0.3 per cent, and in 1213 fatal cases there were 47 of endocarditis, a percentage of 4. Kerr in 171 cases of pneumonia on the autopsy table found 14 instances of endocarditis, *i. e.*, 8.2 per cent. Osler in 100 fatal cases of pneumonias found 16 cases of endocarditis. Sears and Larrabee found 9 in 291 deaths from pneumonia, *i. e.*, 3.1 per cent. Omerod in 183 cases of pneumonia and pleurisy found 6 of endocarditis, *i. e.*, 3.3 per cent. Norris in 500 cases in the Pennsylvania Hospital found 5 of endocarditis. Liebermeister found endocarditis but once in 264 cases treated in the Tübingen clinic, *i. e.*, 0.4 per cent. Sello found only 6 in 750, *i. e.*, 0.8 per cent. Adding these we get 11243 cases of pneumonia with 126 cases of endocarditis, a percentage of 1.1. If the reports based upon autopsy reports are taken we find 1775 cases with 86 instances of endocarditis, a percentage of 4.28. The percentage given by the various authors range from 16 per cent in 100 fatal cases reported by Osler to a minimum of 0.06 per cent by Aufrecht, but it is probable from a consideration the data so far recorded, that the average

is about 1 per cent of all cases and 5 per cent of the fatal cases.

Another question of interest is the relative frequency of endocarditis due to the pneumococcus and that due to other bacteria. Without going into details, I may say that in 944 cases of endocarditis collected from various sources 208 were associated with pneumonia, *i. e.*, 22 per cent. In 127 cases of endocarditis studied bacteriologically 27 were due to the pneumococcus, *i. e.*, 21.2 per cent, only the streptococci and the staphylococci being found more frequently.

It is possible for an endocarditis developing in the course of a pneumonia to be the result of some other organism than the pneumococcus but this rarely happens. Jaccoud in his clinical lectures of 1885-86 reports such a case. In 69 cases in which bacteriological details are given, 64 were due to the pneumococcus only; three times it was associated with the streptococcus, once there was only the streptococcus, and once both streptococcus and staphylococcus.

Both clinicians and pathologists now realize that the words benign and malignant as applied to endocarditis indicate mere difference in intensity and not at all a difference in kind as was thought when these words were originally employed. It is now proven that both sorts of cases are due to the action of bacteria. Either degree of endocarditis may be excited by the pneumococcus and not infrequently we will find one portion of the exudate of the verrucose form while another will be of the polypoid, ulcerative type. Of 39 cases which have been reported

since Netter's review of this subject in 1885, 30 are described as large, polypoid, cauliflower-like, ulcerative; 7 as small and warty and in two cases both forms of exudate were present. From collected data it may be said that the severer form of endocarditis is found in 77 per cent of the cases, the milder in 20 per cent and both together in 3 per cent. In other words, if an endocarditis develops in the course of a pneumonia, it is four to one that it will be of the so-called malignant or ulcerative type.

The following table shows briefly the frequency with which the endocarditis affects the different valves of the heart and compares this frequency with that of endocarditis in general. It is based upon 141 cases of pneumonic endocarditis and the table in Juergensen's article upon endocarditis in Nothnagel's system:

	Pneumonic Endocarditis.	Juergensen's Figures.
Aortic only	39.7%	18.7%
Mitral only	28.3%	66.3%
Aortic and mitral	14.1%	9.2%
Tricuspid only	8.5%	0.4%
Pulmonary only	3.5%	2.3%
Aortic, mitral and tricuspid	3.5%	1.0%
Mitral and tricuspid	1.4%	1.8%

The most striking things about this table are that the pneumococcus is something over twice as apt to involve the aortic valves as the other bacteria and about one-third as apt to involve the mitral, while it attacks the tricuspid twenty times as often.

In 1862 Heschl drew attention by a report of five cases to the frequency with which endocarditis com-

plicating pneumonia is associated with meningitis. Netter fully confirmed this statement, for he found the coincidence of these two complications in no less than 45 of 63 cases in which the records were sufficiently full to supply information upon this point. This is 71.4 per cent. In the 50 cases added to literature since Netter's article, 19 had also a meningitis. Uniting the reports we find the combination in 56.6 per cent of the cases of endocarditis.

Meningitis is a commoner complication than endocarditis, and while approximately 60 per cent of the endocardial cases have meningitis also, only 30 per cent of the latter show endocarditis.

The factors favoring the development of a complicating endocarditis are sex, age, an antecedent endocarditis, alcoholism, debility, anæmia and the other influences generally given as predisposing influences in the causation of complications. A more detailed consideration brings out some interesting facts. In 132 cases there were 92 males and 40 females, respectively 69 and 31 per cent, thus endocarditis complicating pneumonia is about twice as common in men as in women. Since pneumonia is about four times as common in men as in women this means that the female sex is predisposed to the development of the endocarditis.

Consideration of the age of the patients brings out some interesting facts. The following table shows the percentage of cases of endocarditis distributed according to decades and in comparison the age distribution of cases of pneumonia as:

	Pneumococcus Endocarditis.	Pneumonia.
First decade	1.4%	4.76%
Second decade	5.7%	15.6 %
Third decade	10.8%	30.44%
Fourth decade	30.4%	20.75%
Fifth decade	19.5%	15.26%
Sixth decade	21.0%	7.4 %
Seventh decade and over.	10.8%	5.75%

This shows that while 58 per cent of all cases of pneumonia occur during the first three decades, only 17.9 per cent of the cases of pneumococcus endocarditis occur during the same period; that the fourth, fifth and sixth decades supply 35.5 per cent of the pneumonias and 70.9 per cent of the cases of endocarditis. This is in marked contrast to the other infectious diseases which are prone to involve the endocardium. The rheumatism, for instance, causes endocarditis in a large percentage of even mild cases under twenty, while it rather infrequently does so in the adult.

The influence of an antecedent endocarditis is considerable. Thus of the 50 cases published since 1885, 12, *i. e.,* approximately 25 per cent, showed an old endocarditis upon which fresh vegetations had developed. Sears and Larrabee found in their series of pneumonias 80 examples of old endocarditis and that of four of these which went to the autopsy table, three, *i. e.,* 75 per cent, showed a fresh endocarditis.

The cases may be divided clinically into two groups, first, those which are latent and are discovered only at the autopsy table or sometimes months

later when it is found that a valvular lesion is present which did not exist at the time of the pneumonia. This group is and must remain the larger, so many of the cases end fatally before sufficient time elapses to permit of the development of the physical signs upon which alone the diagnosis can be made. The severity of the primary disease often leads to insufficient attention being paid to the other organs, and consequent failure to discover an endocarditis which might otherwise have been recognized. Careful and repeated examinations of the heart should be made in all cases of pneumonia and even more than ordinary care should be exercised in female patients and in those over thirty years of age.

The second group of cases is that in which it is possible to recognize the endocarditis during life. It is not necessary to go into detail as to the symptomatology for the constitutional symptoms and the physical signs of endocarditis due to the pneumococcus do not differ from those of acute endocarditis whatsoever its causal bacterium may be. Temperature is present in practically all cases, though there is here and there a case which, like that reported by Lesage and Pineau, runs an afebrile course. The temperature may continue right on from that of the pneumonia, but in many cases there is a short period during which the temperature is normal. Usually this period is not more than three or four days, but it may be a week or more. The temperature is usually irregular in type, being frequently interrupted by chills, but it may be regularly remittent or intermittent or continuous. In most cases the temperature lasts only a short time, being terminated by

death, but it may continue for weeks, the longest period so far recorded being 180 days.

The physical signs which appear over the heart are determined by the site and character of the resulting lesion. Diastolic murmurs appearing in the course of a pneumonia deserve serious attention, for in 999 out of a 1,000 they are due to organic changes in the valves. Systolic murmurs are worthy of much less serious attention and should be carefully considered before being accepted as evidence of a complicating endocarditis. Their interpretation requires considerable care and sometimes an absolute differentiation between organic and accidental murmurs can be made only after the lapse of days or weeks. The difficulty lies in the fact that the same patient may present an increase of the heart dulness to the right, an accentuation of the second pulmonary tone and a systolic murmur, *i. e.*, all the essential symptoms of a mitral insufficiency, appearing in the course of a disease frequently accompanied by endocarditis and yet be free from any change in the endocardium.

The outcome in cases of this sort is almost universally death.

Cardiac complications, due to myocardial changes, either inflammatory or degenerative in character, are common, and there has been no lack of appreciation of the influence which the condition of the heart muscle has upon the outcome of the disease. Juergensen says the pneumonias die from cardiac insufficiency and Aufrecht affirms that the immediate cause of death in the great majority of cases is a gradual exhaustion of the strength of the heart muscle.

There are a number of factors which work together in pneumonia to cause the cardiac failure. The presence of the infiltrate in the lungs greatly increases the work of the right heart by narrowing the path of the blood through the lungs. At the same time the supply of oxygen is reduced, thus interfering with the aeration of the blood. We find then in the pneumonia a condition which increases the demands upon the heart and at the same time decreases the supply of nutriment. Were these the only factors there should be a direct and simple relation between the extent of the pneumonic area and the degree of cardiac strain, but this is not true, for we often find a marked disproportion between the two. In these cases it is necessary to assume that some third influence is at work and this is the intoxication of the organism as a whole and the heart muscle in particular with the toxins of the pneumococcus. The elevation of the temperature is also an important element in the causation of the cardiac failure, but here again there is often marked disproportion between the height of the fever and the weakness of the heart.

The symptoms pointing to threatening cardiac failure are the same here as they are in other conditions. Marked rapidity of the pulse is a danger signal, but by no means so serious a one as an irregularity. An irregular pulse previous to the crisis is always a cause of anxiety, suggesting, as it does, either an antecedent or beginning myocarditis.

Certain forms of allorrhythmia, such as the pulsus paradoxus, are of no significance when accompanying a pneumonia of the upper lobe. The same is

true of asymmetry of the radial pulses. Dyspnœa or cyanosis out of proportion to the extent of the pneumonia are frequent signs of cardiac weakness.

Examination of the heart itself often shows changes which are suggestive. A moderate enlargement of the heart to the right can hardly be looked upon as a complication, but if it is marked, or if there are other evidences of cardiac weakness, it must be so regarded. Auscultation of the heart aids but little in the recognition of cardiac failure. Accentuation of the second pulmonary tone is frequent and is the expression of the increased blood pressure in the pulmonary system. The degree of this accentuation is dependent upon so many factors, especially upon the condition of conductivity, that no inference can be drawn from the intensity of the tone alone. If, however, during the course of the pneumonia, the accentuation of the second tone decreases markedly without any improvement in the condition of the lungs, it must be regarded as a sign of threatened failure of the right heart.

Another circulatory complication is the phlebitis, which, though rare, is not so uncommon as might be inferred from the fact that it is not mentioned in some of the most extensive articles upon pneumonia. In view of the fact that the fibrin factors are increased in pneumonia we might expect the phlebitis to be a common instead of a rare complication. The rarity is perhaps due to the usually short course of the disease. Sears and Larrabee report 10 cases in 949 pneumonias. Norris found it twice in 500 cases. Miller and Steiner have collected the literature upon these cases and find with their per-

sonal cases forty-eight. The thrombosis usually develops after the crisis or lysis rather than during the course of the disease. It is more common in women than in men and with few exceptions occurs in the lower limbs and more often the left leg than the right. They found the arms involved in but three of the forty-eight cases, but I have personally seen two examples of thrombosis of the upper extremity subsequent to pneumonia. The symptoms do not differ from those caused by thrombosis from any other cause. It is a rather serious affair, for in 34 cases in which Steiner found the outcome recorded, nine died. Of this nine, eight went to autopsy and in five of them the death was due to pulmonary embolism.

Nephritis. When speaking of the symptomatology, mention was made of the great frequency with which albumin and casts are found in the course of this disease. These are not ordinarily to be regarded as evidences of an actual nephritis, but as due rather to degeneration of the renal epithelium. Just where one shall draw the line is difficult to say, but if there is only a degeneration the casts and albumin should disappear within a very short time after the crisis, while if there is a nephritis they will persist.

The frequency with which nephritis occurs is shown by the following table:

	Cases.	Nephritis.	Per Ct.
Vienna	5,738	66	1.2
Stockholm	2,616	52	2.0
Zurich	500	13	2.6
Fraenkel and Reiche...	956	6	0.53

	Cases.	Nephritis.	Per Ct.
Wagner	150	4	2.6
Rosenstein	130	3	1.7
Aufrecht	1,501	16	1.0
Norris	500	6	1.2
Townsend and Coolidge	1,000	9	0.9
Meyer	500	3	0.6

This makes 13,591 cases of pneumonia with 178 cases of acute nephritis, *i. e.,* 1.3 per cent.

The older explanation that the renal changes are due to the temperature must be given up. The current theories are that they are due either to the action of the toxine of the pneumococcus or to the direct action of the cocci excreted through the kidneys. The changes found post mortem exactly resemble those found after intoxication with certain inorganic substances. However, Fraenkel and Reiche found the pneumococcus in the kidneys of 22 of 26 cases examined.

Nephritis may appear at any time during the course of the pneumonia, or it may precede or follow it. It has no manifest effect upon the evolution of the disease nor does it, as a rule, cause any other than urinary symptoms. The urine frequently contains considerable blood, a fact which has led to the idea that there is an inflammation of the glomeruli with degenerative changes in their epithelial covering. Von Kahlden has shown that all or at least the main changes found in these cases are in the epithelium of the convoluted tubules. Uraemia and edema are exceptional.

The prognosis is bad. Nauwerk lost seven of fourteen cases. Of Fraenkel and Reiche's six cases two recovered, one disappeared, and three were dis-

charged with nephritis, from which one died ten months later. The cases usually end in death or recovery, but some become chronic. Leyden, Fraenkel and Reiche, and Eisenlohr each report one instance of the latter.

Other forms of urinary complications are still more rare than the nephritis. I have once observed a very severe and obstinate cystitis in a young girl recovering from a severe pneumonia and pneumococcus endocarditis, but was unable to demonstrate any relation between the pneumococcus infection and the cystitis.

Jaundice. The frequency with which icterus appears in the course of pneumonia is variously stated. This is partly due to the fact that it does not occur with equal frequency in all epidemics, being common in some and rare in others. The great diversity is caused mainly by the amount of attention paid and by the different intensity of the jaundice necessary before note is made of it. The figures run from as low as 0.5 per cent to 28.3 per cent, but most reporters put it at about 1.0 per cent. The following table gives details:

	Cases.	Jaundice.	Per Ct.
Vienna	5,738	30	0.5
Stockholm	2,616	23	0.88
Basel	230	65	28.2
Aufrecht	1,501	15	1.0
Sears and Larrabee	949	29	3.0
Norris	500	18	3.6
Meyer	325	7	2.1
Kerr	171	6	3.5
	12,030	190	1.5

In one report from Basel, jaundice was found in 5.5 per cent of the cases, but in a later list in which particular attention was paid to this point, it was found in 28.3 per cent. The percentage given above, 1.5 per cent, is approximately correct if we include only those cases in which the jaundice is marked and severe enough to constitute an actual complication, but if all grades of jaundice are included, 30 per cent is much more nearly correct.

The pathogenesis of the icterus has been and still is a matter of much dispute and a variety of theories have been advanced, some reasonable and others now entirely abandoned. No doubt there are more ways than one in which the icterus is caused. The old idea of Drasche, Lehman and others, that the jaundice is due to venous stasis with resultant compression of the fine bile ducts has been abandoned for all except a very small number of cases. Jaundice results in this way only when the stasis is great, as it may be in cardiac lesions, but when this explanation holds for a case of pneumonia, it is not this disease, but some persisting heart disturbance which underlies the jaundice.

Another abandoned explanation is that which refers the jaundice to lessened movement of the diaphragm and consequently lessened pressure upon the liver. Were this idea correct jaundice should not occur with pneumonia of the upper lobes, but it does, and it ought also to be disproportionately frequent in cases involving the right lower lobe, which it is not. Petrow studied 67 cases of right-sided pneumonia with especial attention to this point and found only six cases of jaundice, in all of which

there was some lesion of the bile ducts. Moreover if lessened movement of the diaphragm caused jaundice we ought to find it in other cases like pleurisy or paralysis of the diaphragm, but we do not.

Banti, basing his opinion upon 15 cases of pneumonia studied in an epidemic in Florence, believes that the pneumococcus has a haemolytic power, causing so free a destruction of the red blood corpuscles that the bile becomes so abnormally thick that resorption and icterus results. In more than one-half of his cases death occurred and at the autopsy the bile ducts were found to be patent, the bile thick and the fæces dark. Cultures from these cases cause a haemoglobinuria in animals, while the cultures from other cases did not. This would place the jaundice in the haemo-hepatogenous group and make it comparable to the toxic jaundice seen in chloroform, arsenic and phosphorus poisoning.

Other authors, Brislow, Monneret, Jaccoud, Pal believe that some at least of the cases are due to an acute hepatitis as shown by the enlargement of the liver and its tenderness. There is probably not so much difference between this explanation and that of Banti as the words employed might suggest.

In some cases the jaundice is due to a catarrh of the common duct with duodenitis. The causes of such complicating duodenitis are numerous, but it is not likely that many are due to the same organism which causes the pneumonia. In these cases the stools are clay colored, while in the cases previously mentioned the stools are dark, often darker even than normal.

The effect of the jaundice upon the pneumonia is

proportionate to its intensity, and it affects the course of the pneumonia by the influence which it exerts upon the heart, the nervous system, the gastro-intestinal tract and the kidneys. French writers have drawn attention to the fact that jaundice, no matter what its cause is, often excites dilatation of the right heart, even sufficient in degree to render the tricuspid insufficient. It is clear that the addition of a complication, which in itself tends to cause dilatation of the right heart, to a disease characterized by the frequency with which this segment of the heart fails, is a serious matter and renders the outlook less promising. The jaundice affects the sensorium, causes the stupor to appear earlier, lessens the sensibility and excites delirium.

The excretion of the biliary pigments through the kidneys irritates organs already affected by the passage of the pneumotoxines and cocci and may easily convert a kidney which shows mere cloudy swelling and albuminous degeneration into one which is actually inflamed.

Gastro-intestinal disturbances are favored and meteorism is apt to appear. Traube long ago pointed out the fact that distention of the intestines with gas is a serious matter, for it seriously interferes with the breathing in a patient whose breath is already affected. The lack of bile in the intestines favors vomiting and diarrhoea, both serious matters in these cases.

The efforts of some authors to establish a special form of pneumonia under the name of bilious pneumonia seems to me unwarranted. These cases are

due to the action of the pneumococci and the presence of an anomalous symptom does not warrant the creation of a new name. Those cases, where some other organism is at work, should be separated and classified according to the causal organism.

Meningitis. This is one of the commoner and most serious complications although today we cannot agree with Juergensen's statement that death always results. Formerly a clinical diagnosis of meningitis was given up and the case regarded as one of meningismus or meningeal irritation if recovery took place. Today, thanks to the lumbar puncture, we can make a clinical diagnosis of meningitis as positively as it can be made upon the autopsy table and know for certain that recovery is possible.

We must say of this as of all the other complications of pneumonia, the frequency varies from time to time and from place to place. The autopsy records everywhere give a much greater frequency than do the clinical reports, and it is probable that some cases are overlooked in the dead-house because the head and spinal canal are often not opened. The following tables give figures from autopsies and clinics:

AUTOPSIES.

Immerman and Heller..	9	in.	21	cases	= 43.0 %
Osler	8	"	193	"	= 4.2 %
Senger	5	"	165	"	= 3.0 %
Nauwerk	14	"	213	"	= 6.5 %
Bozzolo	38	"	941	"	= 4.0 %
Juergensen	1	"	72	"	= 1.39%
Kerr	9	"	171	"	= 5.2 %

Munich	6 in.	97 cases =	6.1 %
Liege	3 "	42 " =	7.1 %

In 1,915 autopsies meningitis was found 93 times, *i. e.*, 4.85 per cent.

CLINICAL REPORTS.

Sello	5 in	750 cases =	0.6 %
Aufrecht	10 "	1,500 " =	0.6 %
Vienna	8 "	5,738 " =	0.1 %
Stockholm	2 "	2,616 " =	0.1 %
Basel	3 "	230 " =	1.3 %
Nauwerk	14 "	1,172 " =	1.2 %
Biach	15 "	11,422 " =	0.13%
Chomel	4 "	50 " =	0.08%
Sears and Larrabee	7 "	949 " =	0.73%
Norris	4 "	500 " =	0.8 %

This makes 24,927 cases of pneumonia with 72 clinical diagnoses of meningitis, *i. e.*, 0.29 per cent.

There is no doubt that the next few years will see this figure increase up to or possibly even beyond the figures derived from autopsy reports.

No definite knowledge has yet been gained as to the factors determining the localization of the pneumococci in the meninges. Sex has no appreciable influence, according to Netter 90 males to 39 females, about the average distribution of pneumonia according to sex. Age appears here as with the endocarditis and arthritis to have an influence, the third, fourth and fifth decades supplying an altogether undue proportion of the cases. Netter's 124 collected cases were distributed as follows: 26 per cent under 25 years, 44 per cent between 25 and 50 and 30 per cent over 50 years.

Neither the site nor the extent of the pneumonia has any manifest influence. The character of the epidemic, to employ a current expression, has an influence, meningitis being relatively common one year and rare the next. Meyer in Dorpat found five cases of meningitis in eleven autopsies on pneumonias, many times more frequent than the figure given above for autopsy reports.

The meningitis may appear at any time in the pneumonia or it may precede or follow it. About three-fourths of the cases occur during the active stage of the disease, 89 times in 120 cases according to Netter. Nauwerk found the lungs 7 times in stage of red and 19 times in gray or yellow hepatization. Aufrecht gives this table. The meningitis began on the

3rd	day	Once
4th	"	Once
5th	"	Four times
7th	"	Once
8th	"	Twice
9th	"	Three times
10th	"	Six times
12th	"	Twice
17th	"	Twice

The symptoms presented vary greatly, many of the cases being latent. The difficulties in the way of diagnosis are numerous. If only the general symptoms, such as headache, nausea, vomiting, delirium, stupor and the like are present, they are usually referred to the primary disease, which may cause any or all of them. It is only when focal symptoms appear that the possibility of a complicating menin-

gitis occurs to one. Inasmuch as many of the cases die before these develop, many cases are overlooked clinically and must continue to be unless one makes a routine practice of employing the lumbar puncture.

Focal symptoms when they occur are such as are seen in basilar meningitis, whatever its cause. Inequality of the pupils, dilatation or contraction of the pupils, loss of the light reflex, strabismus from paralysis of one or more of the extrinsic ocular muscles with resulting double vision, neuro-retinitis, paralysis of the facial, retraction of the head with rigidity of the neck, a pulse slow out of proportion to the temperature, Cheyne-Stokes breathing, rarely hemiplegia or monoplegia, the Kernig sign. These are not all of equal significance, the rigidity of the neck and the Kernig, for example, being of a low, while a strabismus or a choked disc is of high significance, but the presence of any one or more of these symptoms should lead to the making a lumbar puncture. This will rarely leave any doubt. The only cases in which it will fail are the rare instances in which the meningitis is confined to the convexity of the brain.

The simplicity and the diagnostic value of the lumbar puncture are very great and every one should be prepared to employ it whenever indicated. It is as simple to do as a pleural puncture. The skin of the lumbar region should be carefully prepared and the exploring syringe with a needle at least three inches long should be thoroughly sterilized. The spinous processes of the third and fourth lumbar vertebra are then located and the needle is inserted a half to three-quarters of an inch to one side of the

median line and passed obliquely upward and inward in order to avoid the overlapping of the vertebrae. The patient should be lying upon the side and the back flexed. It is not usually necessary to make lateral flexion as well, but it may be. The needle is passed in until it is felt to enter the canal. Usually the cerebral fluid will escape without aspiration, but if it does not the syringe may be attached and gentle aspiration made. The fluid obtained should be examined for bacteria in both smears and cultures and the character of the cells in the fluid learned. If leucocytes and bacteria are found there can be no further doubt of the diagnosis of meningitis.

The meningitis usually runs a rapidly fatal course, the number of days during which the symptoms lasted being shown in this table from Netter:

1	day	22
2	days	19
3	"	7
4	"	6
5	"	3
6	"	1
7	"	3
8	"	2
9	"	1
11	"	1

Sometimes the symptoms last only a few hours. Recovery is possible and probably occurs more frequently than has been supposed.

Many of the cases of meningitis show other complications also, especially arthritis and endocarditis, the latter being found in about one-third of the cases.

The following case of pneumococcus meningitis, while not an example of one complicating a primary pneumonia may serve to point the description.

The patient, a woman of 43, entered the hospital unable to give any history, such history as was obtained coming from a friend. The illness began three days before admission with a severe frontal headache, with vomiting, which lasted for thirty-six hours. She had several chills during the first night and became so violently delirious that restraint was necessary and the next day she was taken to the Detention Hospital, whence she was transferred to the County Hospital.

Examination showed a well-nourished woman, lying passive and unable to give any account of herself. The elbow, wrist, knee and ankle joints were red, swollen, painful and tender. The heart, lungs, and abdominal organs were negative. The urine contained a trace of albumin with fine and coarse granular casts. The pupils were unequal, the right being the larger. Reaction to light normal. There was rigidity of the neck but there were no paralyses and no Kernig. The pulse was 90, the axillary temperature 99°, the respirations 28. The lumbar puncture gained a diplococcus, answering the description of the pneumococcus.

The next day the pulse was 120, the temperature 100.3° per rectum, the respirations 48. Coma, slight jaundice, no eruption on the skin, bilateral choked disc.

The following day the pulse and temperature records were the same but the respiration had increased to 60. The resonance over both lower lobes

was impaired; the breath sounds were loud and accompanied by a few crepitant râles. The heart was normal in size, the tones pure but a pericardial friction had appeared. There was slight external strabismus, right ptoses; herpes labialis right. The left wrist was punctured and a few drops of thick, white pus obtained and the pneumococci found. The same organism was found in the blood smears.

Autopsy made by Dr. Hektoen showed a purulent pericarditis, pulmonary hypostasis, diffuse purulent cerebro-spinal meningitis, and multiple arthritis. Bacteriological examination showed pneumococci in the blood, pericardium, meninges and joints.

Among the nervous complications there is none more important and more frequent than the delirium tremens. This is so common in alcoholics that it is rare to see even a moderate drinker pass through a pneumonia without delirium, although this is not always violent. The delirium does not differ from that commonly seen in alcoholics. It comes on early, sometimes immediately after the chill but usually on the second or third day. The patients are restless, sleepless, trembling, noisy, and the attention of the physician may be so fully occupied by the delirium that the pneumonia is overlooked, a thing which happens the more easily because the patient is unconscious of any pain in the chest and even the cough is suppressed. This possibility should be kept in mind and all patients with delirium tremens should be frequently examined to determine whether or not a pneumonia has precipitated the alcoholic delirium.

I have already referred to the possibility of pneu-

monia causing a delirium as violent as that of alcoholism and presenting exactly the same clinical characteristics.

A rare complication, to which also reference has been made, is the hemiplegia, monoplegia and aphasia appearing suddenly in the course of a pneumonia and proving by its prompt disappearance that it is not due to any organic lesion. I have never seen but one instance of this. A child of two and a half suddenly presented in the course of an acute pneumonia of the right upper lobe, a paralysis of the left arm. There were no other nervous symptoms, suggesting a meningitis and the fact that the monoplegia completely disappeared in three days excluded any organic lesion. There was no complicating endocarditis.

Neuritis which is a common sequel of many acute infections is rare after pneumonia. There are a few, a very few, cases scattered through literature.

Another rare sequel is the multiple sclerosis, seen rarely after pneumonia as after other acute infectious diseases.

Arthritis. The literature of this subject has recently been collected by Herrick and his conclusions form the basis of this paragraph. Huss regarded acute rheumatism as one of the commonest acute diseases complicating pneumonia and he numbers 22 cases in 2,616 pneumonias, *i. e.*, 0.9 per cent. It is, of course impossible to say how many of these cases were due to the action of the pneumococcus upon the joints but the probabilities are that most of them were. It is certain that the complicating arthritides are more common than one would in-

fer from the fact that Aufrecht does not mention them.

Cave found 2 cases in 2,292 pneumonias. Vogelins found 2 in 3,293 cases. Netter, 6 in 4,156. One reported from Munich in 650 cases and three from Paris in 1,215 cases, Sears and Larrabee 23 times in 949 cases, *i. e.*, 37 in 12,549 cases of pneumonia, a percentage of 0.3. This is probably about the relative frequency of this complication.

Sex and age seem to have some influence in favoring the appearance of arthritis for in 47 cases no less than 40 were males, *i. e.*, 85 per cent. The age distribution was as follows:

Under 10 years	4
10-20 years	0
20-30 years	5
30-40 years	9
40-50 years	15
50-60 years	11
60-70 years	4
70-80 years	2

It is interesting to compare these figures with those given on the age distribution of the endocarditis complicating pneumonia. 17.9 per cent of the cases of endocarditis and 18 per cent of the cases of pneumococcus arthritis occur during the first three decades of life while the next three decades supply 70.9 per cent of the endocarditis and 70 per cent of the arthritis.

The striking similarity of these figures prepares one for the statement that endocarditis and arthritis frequently occur in the same patient.

Trauma has been noted by a number as favoring the development of the arthritis. A previous attack of rheumatism, lead poisoning and alcohol are all supposed to have some predisposing influence.

The arthritis may appear before, during or after the pulmonary process, although in a few instances there was at no time any involvement of the lungs. The arthritis is usually confined to one joint, in 32 of 52 cases. The upper extremity is more commonly affected than the lower, in 23 of 52 cases, in 18 only the lower was involved and in 11 both. Of all the joints the knee is most often affected, it supplying 25 out of a total of 84 joints. Next the shoulder with 12, the elbow with 9, the wrist and sterno-clavicular joint, each with 8, hip and ankle each 3, metacarpo-phalangeal 2.

The joint symptoms are those of an acute inflammation, redness, swelling, pain and tenderness. The character of the fluid can be learned by puncture and at the same time material is gained for the demonstration of the pneumococci, upon which alone an absolutely certain diagnosis can be made.

If the arthritis comes on during the course of pneumonia, there are only the local symptoms but if it begins after the crisis or lysis the temperature goes up for a period and follows usually an irregular course.

The arthritis is not in itself a serious matter but is an evidence of an intense infection and consequently is always a cause for anxiety. Of 52 cases presenting this complication, 32, *i. e.*, 61 per cent, died.

Somewhat more frequently than the arthritis is met simple arthralgia. In these cases there are

no demonstrable changes in the appearance of the joints but they are painful. One or several joints may be so but the monoarthralgia is about twice as common as the polyarthralgia, 35 to 17 according to Netter.

Mention has already been made of the initial or prodromal joint symptoms. I have seen a number of patients entered in the hospital as acute rheumatism, who proved to be croupous pneumonia, and I once saw a patient in consultation on whom a diagnosis of acute articular rheumatism with pericarditis had been made, the joint changes simulating those seen with rheumatism and the consolidation of the pneumonia lying near enough to the heart to be interpreted as a pericardial effusion. The joint symptoms in this as in the other cases quickly disappeared and the clinical course was the one ordinarily shown by croupous pneumonia.

In addition to these complications there are numerous other tissues in which the pneumococci may localize, causing abscesses. It is unnecessary to enter into any detailed description of these for the symptoms do not differ from those caused by any acute inflammation in the same organ. Otitis media, particularly in children, is rather common and is here as elsewhere a serious matter, both because of the danger of the process extending to the meninges and because of the after-effects upon the hearing. The parotid gland is another organ in which the pneumococci settle, reaching it either by extension from the mouth or through the blood. The parotitis may end in resolution, but a considerable

number of the cases end in suppuration. Less frequently similar changes occur in the thyroid.

Suppuration in bursae, in tendon sheaths, in bones and in soft tissue all occur.

Peritonitis may occur secondary to a pneumonia, but it seems best to consider these cases separately.

CHAPTER VII.

PROGNOSIS.

There are a very large number of influences which must be considered in estimating the probable outcome of a pneumonia. Some of these can be measured by a review of long series of cases while others, such, for example, as the virulence of the pneumococcus, are as yet quite imponderable. There can be no question of the fact that this disease, like the other acute infectious diseases, varies in severity from time to time and place to place, and due allowance for the character of the current epidemic must be made. A combination of factors might one year be inevitably followed by death while at some other time they would cause no increase in the anxiety always felt in the presence of so serious a disease as pneumonia.

Variations from the type of the disease should always excite anxiety, for it suggests either that some agent other than the pneumococcus is at work or that the patient is not reacting to the infection as a normal individual should. To be sure, these cases may be favorable, for the variations from the type may be toward mildness as well as toward severity, but any unusual course causes uncertainty and therefore anxiety.

The variation in mortality from year to year has been repeatedly pointed out by numerous authors.

In Stockholm, for example, in 1845 it was 18.4 per cent, in 1851 it was 9.8 per cent and in 1852 it rose to 16.1 per cent. In Vienna the mortality range was much less, from a minimum of 21.4 per cent to a maximum of 26.5 per cent. In Hamburg the figures between 1889 and 1892 ranged from 16.7 per cent to 23.3 per cent. Aufrecht, in his series of 1,501 cases, found the mortality ranging from 31.6 per cent in 1885 and 1886 to 6.6 per cent in 1895 and 1896. The mortality for the year 1894-1895 was 24.9 per cent, and for the entire series 16.8 per cent. According to Townsend and Cooledge the mortality in the Massachusetts General Hospital has risen almost steadily since 1822 from 10 per cent to 28 per cent in 1889. The greatest range is between 1871, when there were no deaths in 18 cases, to 1877, when there were 6 deaths in 11 cases. Put in this way, the statement has little weight, because of the very small number of cases, but if it is said that between 1871 and 1877 the mortality from pneumonia ranged from nothing to 54.3 per cent, it sounds as if it meant a great deal. This will serve as an illustration of the very erroneous impressions one can derive from figures. Edward F. Wells has recently published the following table, which will show in a very large series of cases only a very moderate range of mortality:

	Cases.	Deaths.	Per Cent.
1810-1820.....	338	39	11.5
1820-1830.....	1,035	199	19.2
1830-1840.....	1,062	221	20.8
1840-1850.....	3,265	529	16.2

	Cases.	Deaths.	Per Cent.
1850-1860.....	31,564	6,221	19.1
1860-1870.....	17,519	3,563	20.3
1870-1880.....	10,983	2,053	18.5
1880-1890.....	45,735	8,885	19.4
1890-1900.....	106,898	21,008	19.6

A consideration of the data at hand warrants one in saying that there is a very great range of mortality in short series of cases such as occur in one hospital from year to year, but that in series large enough to counteract the influence of mere chance the mortality does not vary much from time to time.

Sex is very generally stated as having considerable influence upon the mortality and certain statistics bear this out while others contradict it. Juergensen expresses the relative male and female mortality as 2:3.

Thus, for instance, Fränkel and Reiche experience is as follows:

1889	mortality,	males,	19.0%,	females,	5.3%
1890	"	"	15.9%,	"	27.8%
1891	"	"	14.2%,	"	29.2%
1892	"	"	24.0%,	"	20.7%

Geissler had a mortality of 17.9% in 347 men, and 32.5% in 78 women.

In Vienna reports, quoted by Juergensen, the male mortality was 21% and the female 31.1%. While in Stockholm it was 12.9% and 18.6% respectively. Norris gives a male mortality of 30% and a female mortality of 20%. Other reporters find little or no difference. Thus Aufrecht had a mortality of 16.8% in 1,223 men and 18% in 278

women. Sears and Larrabee give the male mortality in their series as 36.5% and the female as 34%. Townsend and Coolidge in a series of 1,000 cases find a male mortality of 25% minus and a female mortality of 25% plus. The 1900 census of the United States records 58,340 male and 47,631 female deaths from pneumonia. In view of the greater frequency of this disease in males, these figures indicate a much higher death rate in women. It is reasonable to assume that these figures are really of more value than hospital reports, for they include the whole country and represent average conditions, which hospital reports certainly do not do. Ziemssen's figures based upon reports from various countries place the female mortality in a number of large cities as follows:

Paris, 48.1%.
Copenhagen, 41.1%.
Hamburg, 43.4%.
Zurich, 51.1%.
Berlin, 43.9%.

Age is a powerful factor in determining the outcome of the case. Very generally it is stated that the mortality of this disease is low in childhood. It should be emphasized that this applies only to the lobar and not at all to the lobular pneumonia which shows a high death rate. The statement should also be modified to the extent that in very young children the cases are apt to end fatally. Fränkel and Reiche lost 9 of 30 cases between 1 and 5 years old, *i. e.*, 30%, and Aufrecht in 57 cases of the same age had a mortality of 24.4%. Aufrecht, however, states that this figure is, in his opinion,

too high, for his experience in private practice has been far more favorable in children of this age. He is unfortunately unable to give definite figures, but his experience is so exactly that of most that all will agree to the statement that except in hospitals and in foundling institutions pneumonia causes only a low mortality in children beyond one year. It is probable that additional details would show that the bulk of the mortality in cases under five as quoted from Fränkel and Reiche, and Aufrecht was from the first and second year. This suggestion is supported by the experience of Meyer, recorded in a review of 500 cases in Mount Sinai Hospital report. He found a mortality under one year of 69.34%, from 1 to 5 years of 21.27%, and 6 to 10 years of 7.89%. From this point up to 20 years of age the mortality is very low, but from this point on it increases as the years go by. The following table is based upon the combined reports of Fränkel and Reiche, Aufrecht, Sears and Larrabee, Norris, Townsend and Coolidge and Meyer.

	Cases.	Deaths.	Percent.
1-10 years	364	58	15.9
10-20 "	925	59	6.3
20-30 "	1,656	224	13.5
30-40 "	1,116	278	24.9
40-50 "	776	263	33.8
50-60 "	369	176	47.7
60-70 "	187	107	57.0
70+ "	87	59	67.8
	5,480	1,224	22.3

The influence of age is more strikingly shown by

the accompanying curve, but neither the foregoing table nor a curve based upon it shows a fact of considerable importance and that is the great

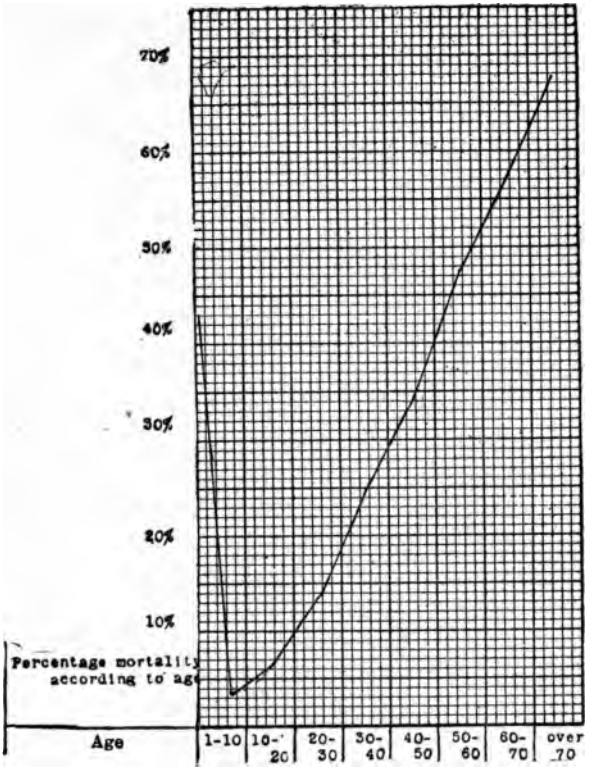


CHART XII. Showing the influence of age upon the mortality.

difference in mortality at different ages under 10. During the first year of life the mortality is high, 43% according to Meyer, and according to Morris

during the first five years of life it is 28.6%, and between 5 and 10 it is but 3.6%. In order to show this the curve for ages under 10 years is drawn both in accordance with the table given and with the figures derived from Meyer, Fränkel and Reiche and Aufrecht.

The influence of the site and extent of the pneumonia upon the mortality has been repeatedly considered, but it is the extent rather than the site which is important. The following table is a combination of the reports of Sears and Larrabee, Townsend and Coolidge, Meyers, Norris and Aufrecht:

	Cases.	Deaths.	Percent.
Right upper lobe.....	448	90	20.0
Right middle lobe.....	95	14	14.7
Right lower lobe.....	1,129	195	17.2
Right lung entire.....	336	141	42.0
Left upper lobe.....	185	34	18.9
Left lower lobe.....	1,002	145	14.4
Left lung entire.....	309	84	27.1
Bilobar	251	91	36.2
Bilateral	307	110	35.5

The following table given by Sears and Larrabee shows clearly the influence of extent:

One lobe, 590 cases, mortality 31.0%
Two lobes, 233 cases, mortality 38.2%
Three lobes, 99 cases, mortality 59.0%
Four lobes, 8 cases, mortality 62.5%
Five lobes, 1 case, mortality 100 %
Double pneumonia, 134 cases, mortality, 42.5%

These figures are higher than those given in the preceding table, but the cases collected by these

authors showed a much higher mortality than is usually observed, namely 35.9%.

It is very generally stated that right sided pneumonias are more dangerous than the left sided cases, and this is borne out by the above. The mortality of the right sided cases being 21.9% in contrast with 17.6% for those in which the left lung is involved.

The value of the temperature in estimating the prognosis is shown by the following table based upon the maximum temperature in 1,515 cases.

Maximum temp.	Cases.	Deaths.	Per-centage.
Under 100°.....	17	6	35.2
100°-102°.....	220	52	23.1
102°-103°.....	302	83	27.4
103°-104°.....	408	109	26.7
104°-105°.....	386	99	26.0
105°-106°.....	148	45	30.5
106° and over.....	34	23	68.0

This shows that the danger is greater if the temperature fails to rise above 100° or passes 105°. And it is also true that the further the temperature is below or above the points indicated the greater the mortality. The cases with very low or no temperature are usually senile. If an old patient shows a temperature of 103° or 104° the outlook is much more hopeful than if it only reaches 101° or 102°, for it indicates that the organism is reacting vigorously to the infection. The table also shows that so long as the temperature ranges between 102° and 105° no inference as to the prognosis can be drawn from it.

In estimating the significance of the pulse rate one must keep several things constantly in mind. A pulse count which would be of serious significance in an adult may mean little in a child. Other things being equal, the pulse will be more rapid in a woman than in a man, in a small individual than in a large, in a nervous person than in one who is phlegmatic. With these things in mind the following table of maximum pulse rates will be of assistance. Cases with a pulse maximum under 120 show a mortality considerably less than the average mortality from pneumonia; those between 120-130 show about the average, and from this point on the mortality rises very rapidly.

Maximum pulse.	Cases.	Deaths.	Per-centage.
Under 100	57	2	3.5
100-110	123	7	5.7
110-120	142	18	12.6
120-130	288	62	21.5
130-140	143	68	47.0
140-150	125	74	59.0
150 and above	83	64	77.0

The character and the rhythm of the pulse must also be considered. An irregular pulse appearing early is always of grave significance and the gravity increases the earlier the irregularity manifests itself.

Something can be learned from the maximum respiratory rate also, but here, too, other factors must be considered, especially the size of the individual and whether or not there is pain on breathing. Rapid breathing in a child or small individual may be expected even when the extent of the

pneumonia is not great. So, also, if breathing excites pain, the respiratory movement will be superficial and an increase in the rate will endeavor to make up for the shallowness.

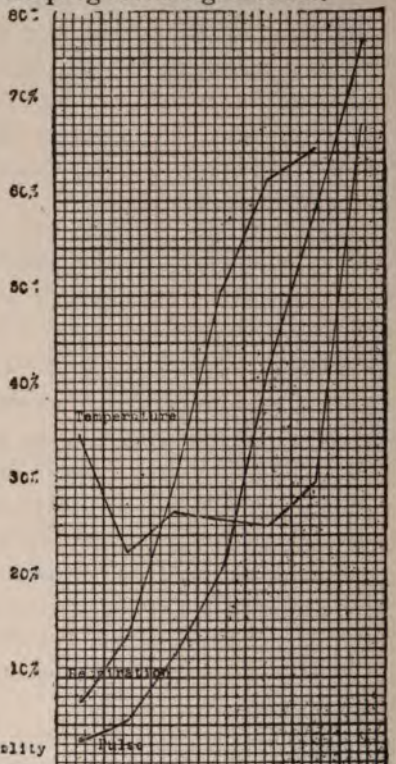
Maximum respiratory rate.	Cases.	Deaths.	Per-centage.
Under 30	77	6	7.7
30-40	257	36	14.0
40-50	384	116	30.2
50-60	123	62	50.4
60-70	98	61	62.2
70 and above	23	14	65.6

The number of leucocytes is of some value in prognosis, for in a general way the cases showing a reasonable increase run a more favorable course than those which do not. There are, however, exceptions to this rule in both ways, *i. e.,* cases with hypo-leucocytosis may end favorably, as those with a marked increase may end in death. If the case in other ways looks favorable, the absence of leucocytosis should not disturb one, but in a doubtful case a low leucocyte count would be an additional disturbing element.

A persistent leucocytosis, after the crisis indicates some complication, even though at the time other signs of such complication are wanting.

Up to recently the presence of the pneumococci in the blood was looked upon as an unfavorable sign, but it can no longer be so regarded. Even when they are so numerous as to admit of easy demonstration in blood smears, they are not a sign of danger.

So far as is yet known, the amount of chlorides in the urine has no prognostic significance.



Maximum temperature	under 100	100-102	102-103	103-104	104-105	105-106	over 106
Maximum pulse	under 100	100-110	110-120	120-130	130-140	140-150	over 150
Maximum respiration	under 30	30-40	40-50	50-60	60-70	over 70	

CHART XIII. Showing the influence of maximum pulse, temperature and respiration upon the mortality.

Several authors, notably Geissler, have attempted to find in the presence or absence of herpes an aid to

the prognosis, holding the herpes to be of favorable significance. I do not, however, believe that they are of significance in as much as they do not occur any more frequently in mild than in severe cases, and are commoner in the young, who show a low mortality rate.

The development of a pneumonia in one suffering from some chronic disease is always unfavorable. Thus a pre-existing nephritis, myocarditis, cirrhosis of the liver, anæmia of any sort make even a very limited pneumonia a very serious affair. The effect of a pre-existing valvular lesion has apparently been exaggerated. Thus, Aufrecht lost 3 out of 13 such cases, and Sears and Larrabee, in a series of 80 such cases, had a mortality of 30.2%, while the death rate in their complete series was 35.9%. Not enough attention has been paid to the site and character of the lesion. Mitral lesions, both the insufficiency and the stenosis, must be more serious as complications, for the pneumonia throws great additional work upon the already strained right ventricle, while aortic lesions, unless secondary to some general disease of the blood vessels, must be looked upon as less serious. One other danger which must be kept in mind in these cases is that of a possible acute exacerbation of the old endocarditis, due to implantation of the pneumococci on the diseased valve.

Inasmuch as the immediate cause of death in the great majority of cases of pneumonia is a general failure of the heart's strength, any evidence of such failure unfavorably influences the prognosis. A considerable increase in the heart dulness to the

right at any time in the course, and particularly so if it appears during the later days of the disease and tends to increase, is a sign of danger. If the accentuation of the second pulmonary tone decreases, without any corresponding improvement in the patient's condition, it has the same significance. Cyanosis or dyspnœa out of proportion to the extent of the pulmonary involvement is also a grave sign. The significance of a very rapid or irregular action of the heart has been referred to.

Chronic alcoholism is always serious, for all experience agrees in finding a much higher mortality in this class of cases than in the average case, and this is true even if the patient shows no nephritis or cardio-vascular disease, and, of course, is still truer if he does. Thus Sears and Larrabee found the mortality in alcoholics 45.5%, while it was 25% in total abstainers and 26.7% in moderate users. Norris had a mortality of 67% in 34 patients known to be drunkards.

The appearance of any of the numerous possible complications during the course of the disease affect the prognosis although not to an equal degree.

The appearance of an edema of the lungs is to be greatly feared, for whether due to a beginning extension of the inflammatory process or to failure of the right heart, the danger to the patient is about the same. Rivalta found a pulmonary edema in 65% of 52 fatal cases. Meyer reports death in 26 of 29 clinical cases.

The pleuritis, even when accompanied by demonstrable amounts of fluid, does not materially increase

the mortality unless the exudate becomes purulent, when the mortality rate is raised somewhat.

Involvement of the other serous membranes is much more serious. The mortality in cases complicated by pericarditis ranges from 50 to 75 per cent. Endocarditis is still more dangerous, the mortality in the cases so far recognized and recorded being over 99%. This, I believe, is much above what it should be, but how much there are as yet no data upon which to base an opinion. Meningitis is almost equally fatal so far as can be judged from the existing reports, but no doubt the general use of the exploratory lumbar puncture will show that more cases of the complicating meningitis recover than has been supposed.

Arthritis also is a serious complication, the mortality according to Herrick being 65 per cent in 52 cases.

Jaundice in the milder grades has no apparent effect upon the mortality, but in those cases in which the jaundice is intense a fatal outcome must be feared. The effects of the jaundice upon the heart, kidneys and gastro-intestinal tract tend to raise the death rate materially, the mortality in these cases being considerably over 50 per cent, ranging indeed in some series of cases near 90 per cent. The cause of the jaundice has some bearing upon the mortality. If it is due to a duodenitis, as shown by symptoms of gastric irritation and acholic stools, the outcome of the pneumonia is much less seriously threatened than in those cases in which there is no obstruction of the common duct but the jaundice is

due to changes in the character of the bile or to obstruction in the intra- or inter-cellular ducts.

Moderate albuminuria even with moderate numbers of casts does not affect the prognosis unless there is reason to believe that the casts are due to antecedent changes in the kidney. Then they are a serious affair.

CHAPTER VIII.

IRREGULAR AND ATYPICAL PNEUMONIA.

There have come down to us a considerable number of terms employed to describe pneumonias which do not follow the usual course or present some peculiarity which has led to their separation into subgroups. Many of these cases are due to the action of the pneumococcus and therefore should be included here, while others of them are due to other organisms and should be sharply separated from the ordinary croupous pneumonia.

Wandering or migratory pneumonia is a form which rapidly extends from one lobe to another, clearing in one as it appears in another.

Massive pneumonias are those in which the amount of exudate in the larger bronchi is sufficient to close the lumen. They do not differ from the ordinary case except in the physical signs. The dulness is more intense, the breath sounds, voice sounds and vocal fremitus are wanting. Usually the bronchial plugs are displaced and expectorated and then the signs seen in the common type are found. During the time when the bronchial tubes are plugged the physical signs are those of a pleurisy with effusion and the exploring syringe may be required to make a differentiation.

Central pneumonias are those which present the

ordinary history of a pneumonia, with the usual rusty sputum and examination of the lungs fails to show any signs which enable one to locate the process. Such a case may pursue the ordinary course without even showing physical signs, but usually as the days go by the process extends to the surface and presents the ordinary signs. In these cases direct auscultation of the chest is of especial value, often enabling one to detect distinct bronchial breathing which cannot be heard by the stethoscope, thus enabling one to locate a pneumonic area which cannot be otherwise detected. The x-ray has been used in these cases and the process located by them.

Pneumonia in children does not present any particular variation from the type. Convulsions, which so frequently mark the onset of acute infections in childhood are not common. Rusty sputum will not be found except in children old enough to expectorate. The most marked difference presented by pneumonia in childhood is its low mortality.

Pneumonia in the aged and in those debilitated by long illness, such as carcinoma, diabetes and the like, is peculiarly insidious in its onset and therefore easily overlooked. There is little or often no rise of temperature, or acceleration of pulse or respiration to warn one and draw attention to the lungs. Cough and expectorate also are often wanting. For this reason, the lungs of such people must be carefully examined whenever they seem to be feeling poorly, no matter how remote the possibility of a pneumonia may seem.

Pneumonia in Alcoholics. Here, too, the onset is insidious and those symptoms which draw attention to the lungs inconspicuous. A delirium tremens is almost certain to develop, and so fully occupies the attention that the underlying pneumonia will be overlooked unless searched for. The lungs should be carefully and repeatedly examined in all cases of delirium tremens.

Abortive pneumonias are those which run their course in a very short time—three, two, or even one day. Such cases, however, are very exceptional and unless the report is complete and accurate, must be looked upon with skepticism. They furnish no basis for the belief, current among some practitioners, that pneumonia can be aborted, a thing as impossible as the abortion of typhoid fever.

Asthenic, toxic or typhoid pneumonias are cases, fortunately rare, in which severe toxic symptoms, prostration, delirium, severe gastro-intestinal symptoms, very rapid and weak pulse, appear early, even before there are any local changes in the lungs. Jaundice is a common symptom and has lead to the use of the term bilious pneumonia. These cases are due to the action of the pneumococcus and they should therefore be included here. They are comparable to the severe asthenic typhoids sometimes seen, and like them are almost always rapidly fatal. They are examples rather of pneumococcus septicæmia than of pneumonia.

Atypical Pneumonias. There occur clinically a considerable number of cases which present many of the features of the croupous pneumonia and yet differ enough from it to have lead many who wrote

of these conditions in the days before bacteriology to separate them and classify them as atypical pneumonias. Here belong many of the instances of contagious pneumonias reported from jails and institutions, and also many of the house epidemics of pneumonia, but there can be no question that such examples of apparent contagion do occur with the pneumococcus pneumonia, but they are rare.

Some of the examples of the asthenic and bilious pneumonias are due to other organisms than the diplococcus of Fraenkel.

Many variations from the average course of pneumonia have been described earlier in the text and so long as the cases can be proven to be due to the pneumococcus they should be included, but when it is shown that the organism at work is some other microbe—the Friedlander bacillus, the staphylococcus, the streptococcus, the bacterium coli communis, the typhoid bacillus, the influenza bacillus—the case should not be looked upon or classified with the croupous pneumonia.

These cases differ not only in their etiology, but in the pathology, symptomatology and course. The changes in the lungs may closely resemble those of the croupous pneumonia, but they are less uniform in appearance and the process is frequently less distinctly lobar. They often resemble an extensive lobular rather than a lobar pneumonia. Microscopically they differ in that the alveoli contain less or no fibrin.

The symptoms differ in many ways. The onset of the rise of temperature may be sudden with a chill or gradual. The course is apt to be irregular

and the duration liable to great variations. The constitutional symptoms are usually more severe. The local signs of the pneumonia are slower in their appearance, are often not strictly lobar in their distribution, and extension of the exudate into the tubules, thus causing the signs of fluid in the pleural cavity rather than those of pneumonia, occurs more frequently than with pneumococcus infection. Jaundice is more common. Splenic tumor is frequent instead of rare. Gastro-intestinal catarrh is not uncommon. The nervous symptoms, especially great prostration, delirium and unconsciousness are frequent. Herpes is exceptional.

Such cases as these last longer and are prone to end in delayed resolution or organization of the exudate. Abscess and gangrene are much more common. The death rate is high.

The diagnosis is based upon bacteriological examination of the sputum and blood.

CHAPTER IX.

PNEUMOCOCCUS INFECTIONS OTHER THAN PULMONARY.

It has been known for years that the pneumococcus is capable of exciting changes in other organs than in the lungs and that while usually such changes were associated with a pneumonia they were not necessarily so. When no pulmonary changes were present there was no clinical method by which such cases could be recognized short of the autopsy table. Post mortem examination showed that the most varied clinical pictures could result from the action of this organism. The case might be clinically a meningitis, an endocarditis, a pleurisy, a peritonitis, an arthritis, a general septicaemia, or other disease, and only after death was the etiological factor demonstrable. Clinically the cases did not differ from the same diseases when excited by other bacteria. It is not here intended to enter into any detailed description of these various processes, but merely to point out the clinical features which these cases present, suggestive of the possible nature of the infection and to outline the method by which the clinical diagnosis can be made as definitely and accurately as the post mortem diagnosis can.

Netter, in 1890, collected 121 autopsies and 31 observations during life of the action of the pneumococcus in order to determine the relative fre-

quency of the various localizations of this bacterium, and in his study found reason for separating in different tables his adult and infantile cases.

The adult table is as follows:

54	lobar pneumonia	= 65.95%
13	broncho-pneumonia	= 15.85%
1	capillary bronchitis	= 1.22%
10	suppurative meningitis	= 11.46%
8	suppurative otitis media	= 9.75%
7	purulent pleuritis	= 8.53%
5	ulcerative endocarditis	= 5.73%
3	suppurative pericarditis	= 3.60%
1	suppurative ostitis	= 1.22%
1	abscess of the liver	= 1.22%
1	inflammatory edema of the leg	= 1.22%

In 82 cases there were 103 localizations of the pneumococcus, and as something like one-fifth of the autopsies were incomplete it is fair to assume that 103 does not represent the full number.

The 54 cases of lobar pneumonia were accompanied by two purulent pleurisies, four cases of otitis media, four of endocarditis, four of meningitis and three of pericarditis.

Four cases of purulent pleuritis were found in which the lungs had never shown any changes.

Of the eight cases of otitis, five were associated with pneumonia, one with meningitis and two were primary.

Seven of the ten cases of meningitis were primary and one case of endocarditis was unaccompanied by a pneumonia.

In the thirty-one cases in childhood the distribu-

tion was different and the important fact demonstrated that while in adults the pneumococci tend to penetrate the deeper air passages, in children it is the tendency to extend upward which is marked. Thus Netter found otitis media 29 times, bronchopneumonia 12 times, meningitis twice, pneumonia, pleurisy, peritonitis and pericarditis each once.

Of thirty-one cases which did not end in death, twenty were purulent pleurisies, five cases of otitis and one each of peritonitis, thoracic phlegmon, abscess of the leg, abscess of the shoulder, mastoiditis and edema of the arm. Only seven of these cases were preceded by pneumonia.

Pearce in 1895 reports a series of autopsies showing much the same facts. His reports show twenty-six cases of acute infections due to the pneumococcus, neither accompanied nor preceded by lobar pneumonia. There were six cases of ulcerative endocarditis, five of meningitis, three of acute pericarditis and six of acute peritonitis. Of these six cases, two were secondary to an acute endometritis, one after a herniotomy, one after a ruptured pyosalpinx, one after a perforation of the rectum and one after an appendicitis. There were also six cases of general infection. From the surgical reports there were twenty-nine examples of pneumococcus infection, fifteen pleurisies, six abscesses, two of otitis, three of mastoiditis, one each of peritonitis, appendicitis and pyosalpinx.

These quotations are sufficient to suggest the wide diversity of processes which may be excited by this most important organism, ranging from a meningitis to an abscess of the leg, from a pyosal-

pinx or appendicitis to an acute endocarditis. And the conditions mentioned above by no means exhaust the list of possibilities.

As stated above, it would carry us too far to enter into any detail in regard to the symptomatology of these widely varying processes and it would be unnecessary, for with few exceptions the symptoms do not differ from those ordinarily seen. There is, however, a group of symptoms constantly recurring in these cases upon which too much stress cannot be laid, for when they are present in their entirety one can with a great degree of probability affirm that the case under consideration is due to the pneumococcus. These symptoms are first a sudden onset with chill and sharp rise of temperature, a leucocytosis and the appearance of herpes, oftenest a herpes labialis. With this trilogy of symptoms one takes little risk in venturing the opinion that the case is due to the pneumococcus and one should always resort to the blood culture to settle the question. The relative value of these three symptoms is not equal. Other infections begin with a chill and this is particularly true of the various conditions listed above. A chill is a part also of the clinical picture of malaria. Leucocytosis also is common to a great many infections, and particularly to the infections with the various pyogenic organisms. The combination then of the chill and leucocytosis is not strongly suggestive of the pneumococcus, but when there is added to these two a herpes, the case is almost proven. A word should be said here of the herpes. These vesicles are so unimportant a part of the disease, and this is especially true when

the symptoms are severe, that they are disregarded by the patient, and too frequently by the physician. Even a single vesicle is to be noted and in many cases the number of vesicles is small. They are often within the borders of nostrils or hidden in the hair of the moustache, so that they are easily overlooked. While the clinical significance is small, their diagnostic significance is very great.

There are other febrile processes in which the herpes labialis is a common phenomenon and some of these present other members of the trilogy mentioned above. The malaria presents the chill and the herpes, but does not show the leucocytosis. The epidemic meningitis shows all three of them, but confusion arises here only when it is a question of whether the meningitis is due to the pneumococcus or to the diplococcus intracellularis. This question, not a very important one in general, can be settled only by lumbar puncture.

It is saying too much to say that these three symptoms warrant an absolute diagnosis, but they should lead one to a bacteriological examination of the blood. The methods of making such examination were referred to on page 87. Too much cannot be said in their praise.

A few examples may serve to point the great diversity of the clinical pictures due to the pneumococcus and to show the value of the blood culture, better than pages of text.

A Mrs. X——, aged 30, seen with Drs. Fowler and Holmes, gave the following history: Thirteen days ago, about five in the evening, in the midst of her usual health, she was taken with a

very severe chill. During the following night she vomited and purged and had a high fever. She was seen by Dr. Fowler the next morning about five. He found her with a temperature of 103° and with negative physical findings, except that during the night she had become intensely jaundiced. Jaundice is really not the correct word, for while the color was that of a deep jaundice, and the urine looked like bile-stained urine, no bile was found in the urine. The patient was taken to the hospital and there it was found that she had a leucocytosis of 58,000. That day she began to bleed from her uterus, which upon examination by Dr. Holmes was found negative. Dr. Holmes decided that there was no evidence of an abortion having been performed and that the infection, manifestly present, was not from the uterus. There was also blood in the urine, in the stools, in the vomitus. The temperature continued high for thirty-six hours, when it returned to normal and remained there. The physical examination remained negative. The leucocytosis persisted. The color mentioned above disappeared at the end of the fourth day.

I saw her upon the thirteenth day. She had by this time become extremely weak and anæmic. She was still vomiting everything taken into the stomach. She was still bleeding from the uterus, bowels and urinary tract. The leucocytosis persisted, though not so high as at first, ranging about 25,000. The physical examination was as negative as it had been throughout, except that below the right angle of the mouth were several herpetic vesicles already a number of days old.

It was easy to say that she had had some infection, but it was not so easy to say what it was. The combination of chill, leucocytosis and herpes suggested the possibility of its being a pneumococcus infection. Blood cultures were made that day by Dr. Rosenow, and pure cultures of this organism were obtained. The patient died a few days later, but no autopsy was permitted.

The case was one of septicæmia, due to the pneumococcus, and while presenting certain symptoms rarely seen as a result of the action of this organism, notably the hemorrhagic diathesis, the group of three symptoms was present.

Another case recently seen with Dr. Kanavel will illustrate another possible combination of symptoms. This patient, also a woman, gave a history of altogether indefinite illness accompanied by chills at irregular intervals, but usually with five to seven days between them. The temperature was very irregular, sometimes high for days, then a period of normal temperature, to be followed by another period of fever. The physical examination was negative, except for a moderate but continuous leucocytosis, averaging 12,000. No more definite diagnosis could be made than that of a cryptogenetic sepsis until cultures were made from the blood by Dr. Koehler. These showed a pure growth of the pneumococci. This case was very different from the other just described, but was due to the same organism.

Some months ago an Italian boy was brought to Wesley Hospital in deep coma, with no history except that he had been feeling poorly for a day or

so, when he suddenly became unconscious some fifteen hours before entrance. The coma was so deep that he could not be aroused. The physical examination was negative, except as showing a rectal temperature of 101.4, respiration rate of 30, and retention of urine. The urine was normal, except for a trace of albumin. The coma continued for three hours, when he regained consciousness, but he was so restless and threw himself about so that it was necessary to fasten him in bed. The next morning the boy complained of headache only. The physical examination was negative, except for a very slight stiffness of the neck. There were no disturbances in the function of any of the cranial nerves, no changes in the retinæ, no Kernig's sign. The temperature was 103° in the axilla, the pulse 100 and the respirations 20. The blood showed a leucocytosis of 36,600 and pneumococci were found in smears and by cultures. Lumbar puncture yielded a milky white fluid, which contained large numbers of the polymorpho-nuclear neutrophile cells. A very prolonged search showed a very few pneumococci. The next day numerous herpetic vesicles appeared on the left cheek and about the left corner of the mouth.

The temperature continued high, the pulse remained slow, generally between 75 and 85 for a temperature of 103°. The physical examination remained negative, none of the ordinary signs of meningitis appearing, nor did any changes in the lungs manifest themselves.

On the eighth day the temperature fell by crisis, and the boy made a complete recovery.

Here, with a totally different picture than that shown by the two women mentioned, we find a leucocytosis and herpes, and while there was no chill the illness began suddenly. This combination of symptoms would suggest a pneumococcus infection, but again it was the blood cultures which settled the question.

Cases of this sort could be multiplied indefinitely, but they serve to illustrate sufficiently the widely diverse clinical pictures resulting from the action of pneumococcus and the great significance of the combination of chill, leucocytosis and herpes. They prove also the great value of the blood culture as an aid to the diagnosis of obscure infections.

In this connection a report of a house epidemic observed by Baduel and Gargani is of interest, both for its bearing upon the diversity of clinical picture resulting from this organism and as another illustration of the value of the blood cultures in diagnosis. They observed eleven cases in one house. The first of these was an otitis media, then came three cases of pneumonia, then four of catarrhal bronchitis, one of which was complicated by a conjunctivitis, then one each of ulcerative gingivitis, parotitis and catarrhal angina. In all of these cases, except the gingivitis, the blood taken from the veins showed the Fraenkel-Weichselbaum diplococcus and in all the blood serum agglutinated the diplococcus.

CHAPTER X.

DIAGNOSIS.

This subject may best be subdivided into three heads—the recognition of the pneumonia, the discovery of the exciting organism, and lastly, the discovery of any complication which may develop.

The diagnosis of a pneumonia is a problem in physical diagnosis and must rest upon physical signs. Previous to the appearance of these, the diagnosis is impossible, although one may be reasonably certain often times from the nature of the onset that a pneumonia is developing. There are not many diseases which begin as suddenly as a pneumonia, and still fewer which begin suddenly with a chill. When, therefore, this mode of onset is present, pneumonia is suggested. If in addition to these the patient experiences a pain in the side, with a troublesome cough with scanty expectorate, the suggestion becomes a probability, but the diagnosis does not become certain until dulness, with the other physical signs of consolidation, appear. If such never appear one must give up a diagnosis which seemed almost certain. When the onset of the disease is not typical, as is often true in old or otherwise debilitated people and in alcoholics, the diagnosis rests solely upon the results of the physical examination of the chest. The same thing is

true of those cases in which some particular symptom, remote from the thorax, draws attention from the lungs. As examples of this may be cited the cases in which the pain is localized over the region of the gall-bladder or the appendix, cases in which there is pain in many joints leading to a diagnosis of gall-stones or appendicitis or acute articular rheumatism and only when the thorax is examined does the true condition present itself. Those who systematically examine the chest in all cases are spared the humility of mistaking a pneumonia for a gall-stone colic or overlooking a pneumonia in some old and feeble patient.

The differentiation of pneumonic consolidation of the lungs from other forms of consolidation is usually easy from a consideration of the other symptoms. Practically the only exception to this rule is the consolidation by compression from a pleural exudate. The physical signs may so closely resemble those of a pneumonia that the exploring syringe is the only means of differentiation. If the pleurisy is due to some organism sufficiently active to cause fever and other constitutional disturbances the resemblance is still greater.

If the efferent bronchus of a pneumonic area is plugged, the physical signs present are exactly the same as those of a pleurisy, except that the pressure effects upon the mediastinum and its viscera and upon the diaphragm are absent. Usually the plug does not remain in place for any great length of time and when it is removed the signs as usually presented by a pneumonia are found. If this does

not happen the exploring syringe alone can make the differentiation.

Having decided that a pneumonia actually exists, effort should be made to determine the bacterium at work. An acute onset with chill, temperature, rusty sputum, leucocytosis and herpes makes it almost certain that the pneumococcus is the offender. The more of these characteristics which are wanting—in other words, the more atypical the onset—the less likely it is that the pneumonia is due to the pneumococcus. On the other hand, no matter how much the case may vary from the type, it is still possible that the Fraenkel-Weichselbaum diplococcus is the cause. The sputum must be examined in all cases, not only for the pneumococci, pus cocci, influenza bacilli and the like, but also and always for the tubercle bacillus.

In practically all cases the sputum contains more than one bacterium and the inference must be that the one present in the greatest number is the one causing the disease. In most cases of croupous pneumonia the diplococcus is found almost pure. A culture made from the blood will settle the question. While the blood cultures are a valuable aid in the diagnosis of the pneumococcus pneumonia, they are indispensable to the recognition of the septicæmias, cases in which the pneumococcus localizes elsewhere than in the lungs.

The detection of the various complications is a matter of attention. They are all accompanied by manifest physical signs and frequent and careful examinations will find most of them. An endocarditis or a pericarditis is rather easily overlooked be-

cause of the pulmonary changes, but many cases discovered on the autopsy table could have been demonstrated clinically if sufficient care had been taken.

Meningitis may be overlooked or may be diagnosed when it does not really exist. Both errors may be avoided by the use of the lumbar puncture, to which reference was made in the paragraphs devoted to meningitis.

An irregular temperature coming on a few days after the temperature has fallen to normal, with a high leucocyte count always suggests a focus of suppuration and because of its great relative frequency, the empyema is the first thing thought of. The chest should be examined for evidences of such complication and the exploring syringe employed repeatedly if necessary. If nothing is found in the chest, the whole body must be searched for signs pointing to some localization of the infection.

If nothing is found, and particularly if the temperature is irregular, with irregular chills, the thought of a possible endocarditis should arise and especial attention be directed to the heart.

If none of these are present the thought of possible beginning organization of the exudate should occur, and careful attention should be given to possible signs of retraction, especially should careful measurements of the thorax be repeatedly made.

CHAPTER XI.

TREATMENT.

There are no questions in the entire range of medicine so difficult to decide as the value of any definite plan or method of treatment of a given disease. So many elements are concerned that it is difficult to reach a just conclusion. The desire to be of assistance to those who are suffering is so strong, that it is often father to the thought that assistance has actually been rendered; and, on the other hand, one must not in the effort to avoid this error go to the opposite extreme. To avoid both the Scylla of optimism and the Charybdis of pessimism is always difficult, but nowhere more difficult than in considering the question of the treatment of pneumonia. There is no disease which attacks more different kinds of people and none in which the natural mortality is more influenced by the character of the patient. A judicious selection of cases will enable one to have a mortality at any desired point between 2 or 3 per cent and 100 per cent. In estimating the value of a given method one must make due allowance for the character of the material. Pneumonia, like the other acute infections, shows also a great diversity in its malignancy. One year the number of fatal cases is low, while the following year, with material of the same sort, treated in the same way, the mortality is high. Why

this is true no one knows, but there is no denying the fact and due allowance must be made for it and any treatment must be tried over several years, and in many places before any conclusions are drawn.

Another difficulty standing in the way of accurate conclusion is that many practitioners do not sufficiently distinguish the various forms of pneumonia nor indeed distinguish sufficiently between pneumonia and other infections of the lungs. The difficulties in the way of accurate diagnosis and of close and continuous watch of the progress of disease in private practice is so great that in the future, as in the past, our conclusions must be based upon hospital statistics rather than upon collected reports from many private practitioners. And in the consideration of conclusions based upon such statistics due allowance must be made for the fact that disease as seen in hospitals is much more severe than as seen in family practice. Hospital patients, generally speaking, are not so well equipped to fight disease. Many of them are alcoholics, are overworked and underfed. Many, too, apply for treatment only after they have been ill for days, and are so sick that it is impossible for them to drive themselves further. The mortality in such cases is, of course, higher than in those who have taken proper care of themselves and are neither soaked with alcohol nor debilitated by exposure and insufficient food. In a general way it may be said that the mortality in private practice is about one-half the hospital mortality—twelve as compared with twenty-five. This means when cases of all sorts and kinds are included, year after year, and for thousands of cases.

Before one can form an opinion of the value of any plan of treatment in pneumonia, one must know first the age of the patients. The importance of this item is shown clearly in Chart XII. One must have details, also, of the habits and physical condition of the patients. There is no better illustration of this than Petrescu's report of a mortality ranging from 1.21% to 2.06% in 2,218 cases treated during a period of four years with large doses of digitalis. This sounds as if the question of the treatment of pneumonia was settled, a large series of cases treated in one way over a period of years with a mortality of less than one-tenth of that ordinarily given. But when it is stated that the patients were all young soldiers, men selected as physically sound and still young enough to be free from the effects of alcoholism, one can only believe that the digitalis had nothing to do with the low mortality. The deaths would have been as few, perhaps even fewer, if they had been given sugar of milk, and it is altogether reasonable to suppose that were the treatment individualized the results would have been still better.

The cases must not be altogether from one or two years, but from several; not from one hospital or city, but from many, and the cases must number not hundreds, but thousands and tens of thousands before one can form any conclusion.

Many plans of treatment have been advocated in the past, and they have ranged from the free and repeated bleeding through many gradations and many drugs to the absolute do-nothingism still prevalent in Vienna. The pneumonias have been

bled and vomited, have had aconite, tartar emetic, large doses of digitalis, large doses of the iodides, pilocarpine, quinine, nitroglycerine, the carbolic acid derivatives, et cetera, et cetera. Each of these has had its turn, each has found its champion who was able to prove to his own perfect satisfaction that a truly valuable treatment had at last been discovered. Many of them have disappeared and after years of abandonment have been again championed. And yet today pneumonia is the despair of the medical profession, constantly increasing in frequency and the mortality today is where it was 200 years ago. Our therapeutics have not kept pace with our increased knowledge of the etiology, pathology and the clinical manifestations of this disease. These, however, are the foundations upon which we must build up a specific method of treatment and it is only through knowledge of them that we can hope to attain the end toward which medicine strives, the cure of the sick. A plan of treatment gained through pure empiricism is not likely to succeed. The chances of failure are too many, indeed, there are a thousand possibilities of failure to one of success.

Theoretically the way in which this disease should be treated is either by destroying the infecting agent or by neutralizing its toxic products. Many efforts have been made, especially by Italian investigators, to find an antipneumonic serum and a considerable number of them have been prepared, but in spite of all the work of Foa and Carbone, the Klemperers, De Renzi, Isabia, Paul, Banti and Pieracciui, Sears and others no certain progress has been made. Some

investigators claim encouraging results; others working with the same serum have failed completely. The clinical results have been in no degree comparable to the laboratory experiments. Any opinion of the value of the serums must be suspended until more data have been accumulated, and it is not probable that any of the serums so far employed will be ultimately established in favor.

The article of Aufrecht in the Nothnagel System has reawakened an interest in the use of quinine in pneumonia. Years ago this drug was very generally used, but the results were not sufficiently definite to gain for it a permanent place and it disappeared to reappear under the influence of Aufrecht's example. He uses three doses each day of one gram of the neutral tannate of quinine combined with an equal amount of the saccharated oxide of iron. His experience differs from that of others, notably Sée and Juergensen, who observed considerable reduction of temperature after doses of 30 grains given at night. Aufrecht has not noted any antipyretic effect and expresses the opinion that the beneficial effects of the quinine are due to some specific action which it has upon the pneumococcus or its toxines. In cases in which there is delirium and in which the pulse is small, soft and rapid and the disease is at its height, and earlier in severe cases, he gives the quinine hypodermically, using a watery solution of the hydrochlorite of quinine, one-half gram twice daily for two days, or sometimes he skips a day. The results which have lead to his support of the use of quinine are 121 cases in two years with an average mortality of 7.4 per cent.

The mortality the first year was 6.6 per cent and the second year 8.2 per cent.

It is difficult for one who is at all conservative to share his encouragement. His experience is too small and Aufrecht himself has had nearly as favorable results in other years with a purely expectant treatment, *i. e.*, 9.8% in 1880. It is difficult to believe that 45 grains of the tannate of quinine is going to have much effect upon such a disease as pneumonia unless the effect were truly specific as is that of the quinine upon malaria, and were the effect so specific, the drug would never have fallen into disuse.

Altshul has recently appeared as an advocate of the use of the iodides, long ago employed by Semmola and Ferrara, but he uses them in much larger doses. They used 5 to 10 drops of a saturated solution of potassium iodide every two hours during the first twenty-four. They claimed a marked reduction in the mortality, but admitted that the iodides were useless if begun later than twenty-four hours after the onset of the disease. Velten some years later urged their use, but employed 90 grains during the first twenty-four hours. He also was enthusiastic, but failed to give details. Altshul uses yet larger doses, 10-20 grains every two hours, and has given as high as 1,500 grains per day. He quotes Dr. Prentiss, of Holyoke, Mass., as having given 3,000 grains per day for four days. Any one who has ever given as much as 1,000 grains of the iodide of potash to a patient would feel strongly inclined to question the possibility of giving so large an amount as 3,000 grains to any one, and admitting

the possibility, one would still question its advisability. There, as with the quinine, one can say that if the iodides had much effect they would never have been abandoned.

Pilocarpine in doses sufficient to produce profuse sweating has been urged and employed as a means of treatment, and upon the basis of a low mortality in small series of cases has been advanced as a specific. It, too, has been abandoned. Recently Rochester, of Buffalo, has advocated the use of sweating by means of hot mustard foot baths, given in bed. He believes that the toxines are removed in this way, but when one recalls Bouchard's estimation that 100 litres of perspiration contain less of toxines than 32 grams of blood one cannot believe that sweating a pneumonia is going to remove much toxic material. Venesection would do much more in this way. Rochester's report shows a low mortality in hospital material, but I believe that the results are due rather to an intelligent individualization of his treatment to fit the case than to the sweating to which all were subjected.

Aconite and veratrum have both been urged, particularly as a means of aborting the pneumonia. They still find their advocates, but there is no authority known to me who regards them highly. Personally I have never employed them because some years ago I ran a series of some seventy odd cases parallel with a colleague who employed the aconite in an equal number of cases, while I did not. The aconite had no effect either in shortening the course or rendering the patient more comfortable,

or in lowering the mortality, and I have therefore always looked upon it as useless.

A large number of practitioners have advocated the use of various antiseptics, notably the salicylates, oil of wintergreen, and of late particularly creosote and its various derivatives, guaiacol, carbonate of guaiacol, carbonate of creosote, thiacol. These men also find support for their opinion in statistics.

Venesection, for many years the main stay in the treatment of pneumonia, was long ago discontinued, and no one since the article of Dietl in 1849 has advocated its employment as a routine measure. The reaction against blood-letting was so great that for many years it was discontinued entirely, an extreme which was as unreasonable as the other, though less dangerous. I am glad to be able to say that venesection is again being employed and under the circumstances in which it is indicated there is no therapeutic measure of greater value. The cases in which it is proper to employ it are not numerous, and they are of such a character that the mortality must from the nature of things be high.

The use of digitalis is also an old method of treatment which, like others, has been employed, then dropped, and then revived. Petrescu in 1888 appeared as an enthusiastic advocate, using in twenty-four hours an infusion of 12 grams to 200. His mortality was very low, but as his cases were all in young and healthy adults his report did not lead to the adoption of this method as a routine treatment, and as the years have gone by we have heard less

and less of the employment of large doses of digitalis in the treatment of this disease.

The following quotation from Pel is inserted both because of its bearing upon the use of digitalis and also because it applies with equal force to other methods. Similar remarks could be made of different individuals. Some are easily excited to enthusiasm, others are cold and only aroused by very positive and unquestionable evidence. Pel says: "National peculiarities appear to have some part in this discussion. The more warmly and ardently the blood flows through the veins, the greater the enthusiasm with which digitalis in large doses is regarded. Under their clear blue sky, the Italians regard it as a specific against the toxines circulating in the blood. In Roumania, with its oriental cast of mind, the home of this method, it is regarded with fatherly love. In France, where the blood is still more easily fired, it finds warm supporters. In Vienna, the number of enthusiasts decreases, but there are still a few. In North Germany one must hunt for its apostles with a lantern, and in cool, phlegmatic Holland, the method finds no supporters, but meets with active opposition."

The above paragraphs do not by any means exhaust the list of methods which have been tried and are still tried to combat this disease, but to-day the most authorities unite in avoiding them and in treating each case individually and thoughtfully rather than in any routine way. Recognizing that we as yet possess no method which can be regarded as even in a minimal degree a specific, every effort is made to support the patient in his struggle and to avoid all things which can injure him.

The treatment of any disease against which we have a specific remedy requires only a very ordinary intelligence, but when the physician is called to combat such a disease as pneumonia, against which no specific exists, the very greatest knowledge, attention, skill and experience is required. To have no specific treatment does not mean that we must sit idly by doing nothing. Just the opposite is required, active, constant and thoughtful attention, but doing no one thing for the sake of doing something. The attitude of the physician should be one of "watchful expectancy," ready at any moment to assist, but so long as things are going well, content to leave well enough alone.

How, then, shall the cases be treated? What can be done to enable the patient to best resist the infection?

The patient should be placed in as large and airy a room as is available. In view of the lessened breathing surface, an abundance of fresh air should be supplied. This is best supplied by securing free circulation of air through one, or, better, two windows, kept constantly open. The number of attendants should be one usually, and very rarely more than two. Except for these the patient should be left alone. The temperature of the room should range about 65°, better less than more than this.

The bed should be a single bed, with firm springs and mattress. The clothing should be light in weight, and the amount of it must be proportionate to the temperature of the room. The bed clothing of the patient should be made in such a way

as to permit of its removal without raising or disturbing the patient. The garment which seems best to me is a short shirt, with loose sleeves and open down the back. It may be of cotton or woolen. The cotton batting jacket, so long popular, has no advantages, and is usually a very dirty and cumbersome garment. The reasons for using such a bed and bed-clothing are obvious, and any mention of them might be omitted were it not for the fact that one frequently sees pneumonic cases in wide beds, which make it difficult to handle them, and in bed-clothing which cannot be changed without greatly disturbing the patient.

The diet should be such as is appropriate to a patient with temperature and in bed. Milk, milk with lime water, peptonized milk, malted milk, buttermilk, koumyss, ice-cream, cereals of all sorts, eggs in various ways, soft, raw or in the shape of an eggnog or egg and sherry, broths, gruels, fruit juices, custards, beef juice, scraped beef. Foods of this sort in moderate quantities and at short intervals.

Water should be given freely, not waiting for the patient to ask for it, but offering it and insisting upon it at least every two hours. At least three pints of water should be taken daily.

The use of alcohol, both as a food and as a stimulant, has been and is a matter of much discussion and seems to be largely a matter of opinion. Some use it always; some never use it. Personally, I see no particular advantage in its use, except with patients who have been accustomed to it. It should then be used in amounts proportionate to the hab-

its of the patient. To give alcoholics freely to those unaccustomed to their use is decidedly harmful. In moderate quantities it is a convenient and useful stimulant, and in the shape of an eggnog or whiskey toddy is a very acceptable addition to the dietary.

Care must be taken to secure for the patient a proper amount of sleep, often a difficult thing in a patient suffering from dyspnœa, cough and pain. Most patients, however, can sleep if left alone in a quiet and darkened room. During the night hours the patient should not be fussed over any more than is necessary, and definite instructions should be given to secure this quiet. If such simple means as these fail to secure sleep, a whisky toddy, a dose of chloral, trional or some other drug of this class may be cautiously employed.

The bowels should be kept open and it is always well to prescribe at the first visit a dose of calomel or some other drug of this class to secure a free evacuation of the bowels.

The skin should be kept active by frequent tepid bathings and alcohol rubs.

In the great majority of cases this is all that need be done for the patient, and only when some particular symptom or symptoms are excessively marked is further intervention required.

Fever. The temperature to employ antipyretic measures is strong, especially when the temperature is averaging above 103°, but a consideration of the mortality rate in large series of cases shows that the temperature is not to be feared, unless it passes 105°. As stated earlier, the mortality in pneumonia averages the same for all cases in

which the maximum temperature lies between 100° and 105°. Since this is true, it is clear that there is no justification for actively combating the elevation of temperature. The only reservation which need be made is that in people beyond the mid-point of life the temperature averages lower than in the healthy adult, and that, therefore, the point for active interference in such cases had better be 103° than 105°.

Below this point the only measure needed is frequent tepid or cool sponging. These are employed for the comfort which they give the patient and for the influence which such bathing has upon the skin.

When more active means are required, the best is the cold tub bath or pack. These are very disagreeable to many patients and when so it is best to put the patient in a bath at 90° F. and then cool the water to 80° or 75°.

I am very strongly opposed to the use of the numerous antipyretic drugs now on the market for the purpose of reducing the temperature. They are altogether too dangerous—far more so than the temperature for which they are employed. Quinine has been employed by some, notably by Juergensen, for this purpose, and was highly regarded by him. He employed doses of 30 grains in the evening. The effect came slowly and was maintained so that it was not necessary to repeat the dose oftener than once in 48 hours. Others have failed to get any such effects.

Pain. The pain in the side is often a very distressing symptom requiring relief, and any one of

a number of simple methods may be employed, the particular one selected being determined by the effect. Many patients are relieved by the ice-bag or by cold applications kept continuously over the painful area. Of these two, the bag is the preferable inasmuch as it does not require frequent changing and consequent disturbance of the patient. Other patients experience no relief from the cold, but do from hot applications, which should then be employed. The hot water bag is the most convenient, but poultices or cloths wrung from hot water serve equally well. The various pastes now extensively advertised have no advantage over other means of applying heat and are dirty and therefore disagreeable things to have about. They have no virtue except that they are harmless, and if the mental equipment of the patient or the patient's family is such that an appearance of activity must be maintained, they are convenient.

If neither heat nor cold gives the desired relief, counter-irritation by means of iodine or a mustard plaster may do so. These are often very effective, but they should not be continued to the point of blistering.

Leeches and cupping may be employed for this same purpose, often with very great satisfaction to the patient.

Another simple procedure, often helpful and at the same time harmless, is strapping the chest with adhesive strips just as is done for broken ribs or for pleurisy. The strips should be about an inch and a half wide and long enough to pass beyond the median line, both front and back. The lowest

strip should be applied first at the level of the twelfth rib, each succeeding strip overlapping the one below it. They should be carried up until the shoulder-chest muscles prevent further application. Strips should be then applied from before, backward over the shoulder. This fixes the entire side and greatly relieves the pain without increasing the dyspnœa.

If these means fail, then morphine should be given hypodermically in doses sufficient to render the pain endurable, but not enough to cause its complete disappearance. The percentage of cases in which it must be employed is small, but when required it can be used with perfect safety and perfect satisfaction.

Cough. This is always a distressing symptom, but not usually sufficiently so to require sedatives, and these should be avoided if possible. Only when the persistent cough is robbing the patient of all rest and exhausting him should morphine or heroin be used. In certain nervous patients the cough can be controlled to some degree by conscious effort on their part—not to yield to every stimulus to cough. Of course, the cough cannot be suppressed entirely, but it can be considerably limited.

During the early stages of the disease, before the exudate has begun to soften, it is useless to give the various expectorant mixtures one sees so frequently employed. Not only do they accomplish no good, but they are potent for evil, causing nausea and disturbing the digestive processes, already enfeebled by the fever. Later, the common ammonium cough mixtures are useful, aiding the patient in the removal of the softened exudate. For

a long time, however, I have personally ceased to use them, employing in their place the carbonate of guaiacol in five grain doses either with or without small amounts of apomorphia, 1-20 of a grain. I was led to this by the greater efficiency of the guaiacol in the treatment of acute bronchitis and have been satisfied with the results.

Dyspnœa. The degree of this symptom varies greatly, but fortunately in most cases it is not so extreme as to require special effort on the part of the physician. If the dyspnœa is due to the fact that the pulmonary involvement is extensive not much can be done. When such cases are seen early, that is, during the first forty-eight hours, before hepatization of the lobes is complete, nothing is so efficient as a free blood-letting. The sense of comfort and the relief from pain and dyspnœa experienced by a full-blooded and robust adult after the withdrawal of 18 to 20 ounces of blood is so great that it is easy to understand the enthusiasm of our forefathers over this method. If the patient is weak and anæmic, or if hepatization is already complete, no such effects are seen and venesection should not be employed.

Inhalation of oxygen in these cases is of very doubtful value as in all other circumstances where the cause of the dyspnœa is a reduction in the breathing surface. After a very considerable experience in the use of oxygen, I can say that I have never yet seen a single instance in which it appeared to me likely that the use of oxygen had any effect on the outcome of the disease. Its use is harmless and in as much as some patients claim to experience relief from the inhalations they may be tried and if liked

by the patient, continued. The use of oxygen appeals to the limited knowledge of physiology possessed by the average layman and as a consequence there is a wide-spread prejudice in favor of the use of oxygen in pneumonia. In as much as its use is harmless, it is easier to yield than to argue and the oxygen tank will continue to appear at the bed side.

In using oxygen the cone usually supplied with the tank should not be held over the nose and mouth, like an ether cone, but at a distance from them, spraying the oxygen over the face.

Cardiac Failure. Death is probably more frequently due to cardiac failure than to any other one cause and for this reason the circulatory conditions must be anxiously watched. Undue rapidity of the pulse and even more irregularity of the pulse must be attended to at once, as must also increasing dulness of the heart to the right, decreasing accentuation of the second pulmonary tone and cyanosis, especially when out of proportion to the extent of the pulmonary infiltration.

In many cases, an ice bag kept constantly over the precordial region will be found sufficient, but when it is not the usual cardiac stimulants must be employed. Of these the best is, in my opinion, digitalis, using an infusion, a tincture or fat-free tincture or the powdered leaves. Each of these has its adherents. The various alkaloids of digitalis may be used. Of these the digitaline has never given me any satisfaction, but the digitoxin has proven of the greatest value. The dose of 1-250 of a grain as ordinarily employed, is, I believe, too large and a dose of 1-500 of a grain will be found more satisfac-

tory. The drug is one of great power and must be constantly watched to avoid causing too much slowing of the heart or vomiting.

Many prefer strychnia as a stimulant, and it certainly has some advantages, especially the ease with which it can be given hypodermically. Others prefer caffeine, camphorated oil or ammonia. The various alcoholic beverages are useful as stimulants, especially in those cases where only a little is required.

Definite directions as to the doses in which these various cardiac stimulants should be employed cannot be given for each case must be treated according to symptoms which it presents and the amount of stimulation must be varied from day to day and even from hour to hour.

Cases in which an old valvular lesion exists had best receive small doses of digitalis from the onset, just as those presenting evidences of chronic myocarditis or arterio-sclerosis should be put and kept under the influence of nitroglycerine.

If, in spite of stimulation, the evidences of cardiac failure increase, or if signs of pulmonary edema supervene resort to venesection is advisable. One should, however, realize that such cases are desperate and not expect too much from the bleeding. In a certain, though a low percentage of cases, this procedure will be life saving.

Nervous Symptoms requiring treatment are not numerous, and of them the delirium is most common. In the milder degree the presence and influence of an efficient nurse is all that is required. Others require the continuous use of an ice cap, simple

but often effective. Some of these cases are wonderfully benefited by a general cold bath. If all of these fail, and they often do, especially in the alcoholic delirium, chloral hydrate in doses up to 60 grains per day will be found generally efficacious.

Sleeplessness can generally be combated by securing quiet for the patient, but if it persists it may seriously injure the patient and should be controlled, preferably by a small dose of morphine.

The gastro-intestinal symptoms rarely require any attention beyond the initial cleaning out of the bowels by a moderate dose of calomel, followed by a saline. If the bowel movements are unsatisfactory, mild laxatives or an enema may be employed. Fermentation in the intestines, if in any way marked, is worthy of attention, not only because of the possible intoxication occurring with it, but especially because the intestines distended by gas, interfere with the movement of the diaphragm. It should be checked by some one of the numerous intestinal antiseptics, salol, guaiacol carbonate, acetozone, etc., and all milk should be withdrawn from the dietary for a period of at least twenty-four hours.

Jaundice does not require any special treatment except that, when present, fermentation of the intestines is often excessive, and it is well to employ some intestinal antiseptic.

Albuminuria. The great frequency, almost constancy, with which albumin and casts are found in the urine shows that the disease itself causes considerable irritation of the kidneys and for this reason care should be taken to avoid both in diet and in drugs all bodies which in their excretion irritate

these organs. The use of urotropine in these cases is not indicated as in certain other infections, notably typhoid and scarlet fever, for the irritant seems to be toxic rather than infectious. Other than care of the diet and the free ingestion of water, the kidneys require no attention.

Complications. Many of these, such as the meningitis, endocarditis, and the like, need no discussion here, for they must be handled in exactly the same way in which such conditions are treated under other circumstances. The same may be said of the empyema and purulent pericarditis. When recognized they must be drained, for while it cannot be denied that exceptionally patients recover from these without operation, the chances of spontaneous recovery are too few and the dangers too many to omit drainage.

When resolution is delayed, the patient should, if possible, be removed to some locality where he can live constantly out of doors, and the internal administration of the iodides should be started. Massage of the chest and breathing exercises should be instituted and continued until the lung is again clear.

The same things are even more imperatively indicated if instead of merely delayed resolution, organization of the exudate takes place.

Abscesses of the lung should be treated by drainage if they can be located with sufficient accuracy to indicate to the surgeon where he shall operate. If this cannot be done, the general health of the patient should be improved by every dietetic and hygienic means possible in hopes that nature may heal the abscess without artificial drainage. Fortunately

this happens not so infrequently, although recovery is often much delayed. If gangrene of the lungs results from the pneumonia, drainage should be established if localization of the gangrenous focus is possible. If it is not one must do the best he can by general hygienic measures and by inhalations of turpentine, eucalyptus, creasote and the like.